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Table of Contents

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SOME CLINICAL OBSERVATIONS ON BLOOD PRESSURE AND THEIR PRACTICAL APPLICATION, WITH SPECIAL REFERENCE TO VARIATION OF BLOOD PRESSURE READINGS IN THE TWO ARMS.[1]

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THE presentation of a paper on blood pressure at a time such as the present when the medical Press has been literally inundated with contributions on this subject for some years past would seem rather superfluous. This surmise is apparently confirmed when one considers the frequency with which blood pressure is discussed over the air, in the lay Press, in the deluge of advertisements for patent medicines, at ladies' afternoon tea parties, and worst of all when the patient presents himself or herself to the medical attendant with the announcement that he or she is suffering from blood pressure.

This complaint is certainly the prevailing vogue with the laity of the present day. However, the very multiplicity of works, with their extreme divergence of opinion on various aspects of the subject, would indicate at once a lack of unanimity, and therefore a necessity for further research.

I hope to present tonight the results of some clinical observations, made in the course of practice, which I trust will be of interest to members, and stimulate thought and discussion. I do not wish to take up valuable time with a minute consideration of the fascinating physiological aspect of the subject, except to mention in passing that various investigations with a great variety of instruments, on both human beings and animals, have confirmed the fact that the present day methods of estimating blood pressure with either the mercurial or aneroid type of instrument give quite reliable figures for all clinical purposes.

STANDARD OF NORMAL PRESSURES.

With most of the investigations that are carried out during the ordinary clinical examination of the patient at the bedside or in the consulting room,

[1] Read at a meeting of the Victorian Branch of the British Medical Association on October 2, 1935.

there is a very definite standard of normality, any variation from which is at once an indication of some departure from the conditions which should apply in normal health, for example, temperature, pulse rate, respiration rate, urinary findings *et cetera*. In dealing with blood pressure readings, however, there is a totally different state of affairs from the clinician's point of view.

In an endeavour to arrive at an approximate standard of normality for practical use, a number of prominent insurance companies have been approached, as these institutions seemed to be the most favourably situated for the collection of records of normally healthy members of the community.

In the first place, all the recognized tables of normal pressures have been derived from either American or British figures. Secondly, each company has its own standards, often guided by the clinical experience of its medical officers, and therefore necessarily varying with individual companies. This applies particularly to the actual technique of estimation of the pressure, which will be dealt with in more detail later.

The outcome of these inquiries, however, reveals the fact that the recognized normal pressures for the various age groups, especially in the later years of life, are considerably lower than what one might regard as the popularly accepted figures in general practice. I refer here to the traditional rule of 100 *plus* the age up to 150 or 160 millimetres of mercury for the systolic pressure, which seems to be the working rule for most practitioners.

Further, very little reference is made usually to the diastolic pressure, and yet from the point of view of prognosis this reading would appear to be of even greater value than the systolic. This will also be amplified later.

We must bear in mind, however, that we are dealing, in practice, with people who have come to consult us for some condition of impairment to health; and that we are not, therefore, handling individuals who could be regarded as suitable for the preparation of normal standards.

Reference to Tables I and II will demonstrate this point, and the view of the insurance companies is borne out by the increased mortality rates in individuals whose pressure is higher than the recognized normal, as indicated in the latter table supplied by one of the companies.

Table I.

Standard Normal Readings of Six Companies.
(Pressure stated in millimetres of mercury.)

Age in Years.	Systolic.						Diastolic.						Pulse Pressure.					
	1	2	3	4	5	6	1	2	3	4	5	6	1	2	3	4	5	6
20	123	120	120	120	120	125	80	80	80	80	79	—	43	40	40	40	41	—
25	125	122	—	—	121	—	81	81	—	—	80	—	44	41	—	—	41	—
30	126	123	123	130	122	130	82	82	82	90	81	—	44	41	41	40	41	—
35	127	124	—	—	123	—	83	83	—	—	82	—	44	41	—	—	41	—
40	128	126	126	140	125	135	84	84	84	90	83	—	44	42	42	50	42	—
45	129	128	—	—	127	—	85	85	—	—	84	—	44	43	—	—	43	—
50	131	130	130	150	129	140	86	86	86	100	85	—	45	44	44	50	44	—
55	133	132	—	—	131	—	87	87	—	—	86	—	46	45	—	—	45	—
60	135	135	135	155	134	145	89	89	90	100	87	—	46	46	45	55	47	—

Method of Estimation of the Pressure.

The method of estimation of the pressure is an important factor in compiling any standard of normal pressures, since the reading may vary according to the instrument used, the details of technique as to the actual estimation and the criteria for determining the point of recording the systolic and diastolic pressures.

Table II.

Increased Mortality with Pressures (Millimetres of Mercury) above the Normal.
(Quoted by one of the companies.)

Ages.	Average Systolic Pressure.	Over Average. (X.)	High. (Y.)	Highest. (Z.)
15 to 29	120	125 to 135	136 to 144	145+
30 to 44 ..	125	130 to 140	141 to 149	150+
45 to 53	130	135 to 145	146 to 154	155+
54 to 70 ..	135	140 to 150	151 to 159	160+

TABLE IIA.

Group.	Exposed to Risk.	Actual Deaths.	Expected Deaths.	Percentage of Expected Deaths.
(X) over average	34,555	371	242	153
(Y) high	25,570	382	212	181
(Z) highest ..	46,750	750	348	215

For an equitable comparison of any group of figures it is obvious that some uniform routine of estimating the pressure must be adopted in each instance. That this is essential is shown by the degree of variation in the following data supplied by some of the insurance companies with reference to the technique of the estimation.

Company Number 1.—No uniform position of the candidate is adopted, and the diastolic pressure is recorded just at the point at which all sounds disappear.

Company Number 2.—The candidate is examined sitting; no definite standard of pressures is adopted. The final decision rests with the chief medical officer. "Silence to silence" criteria for systolic and diastolic readings are adopted.

Company Number 3.—No position of the candidate is specified. "Silence to silence" method is used. All pressures are queried when the figures are above 145 millimetres for the systolic and when the diastolic (fifth phase) is above 90 millimetres, irrespective of age. Diastolic pressure is the more important figure. Pulse pressure should be about 35 millimetres.

Company Number 4.—No standard method or position of the examinee is adopted. There is no standard scale of blood pressures according to age, height, or weight. The diastolic reading is taken at the commencement of the fourth phase.

Company Number 5.—Recumbent position on the couch is adopted, and the test is made towards the end of the examination. "Silence to silence" method is used. There is a recognized standard scale of pressures.

Company Number 6.—No standard position of the candidate and no recognized normal scale are used. Reliance is principally placed on the systolic reading.

Company Number 7.—The examinee is recumbent. A recognized standard of normal pressures is adopted. The diastolic pressure is read at the commencement of the fourth phase.

Company Number 8.—No position of the candidate is specified. "Silence to silence" method is used. The diastolic pressure is regarded as the more important. The final decision rests with the senior medical officer.

Let us turn for a moment to the various sound phases which are heard during blood pressure estimations.

After the cuff has been inflated until all sounds over the artery have been obliterated and the pressure is gradually lowered, we hear:

1. A sharp distinct systolic tapping sound described as a tap, click, or pop.

2. As the pressure is lowered this is followed by a less sharp sound, more in the nature of a murmur, which may become so faint as to be imperceptible.

3. This is followed as the pressure is further released by a reappearance of the sharp distinct sound, possibly not so loud, and often described as a thud.

4. The next phase begins just as this sound becomes more dull.

5. The fifth phase as the sound disappears altogether.

This can be well demonstrated diagrammatically as in Figure I.

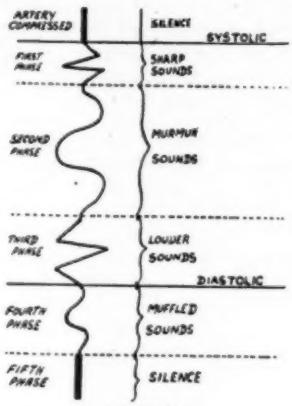

FIGURE I.

Most authorities are quite definite as to the point of determining the systolic reading, that is at the commencement of the first phase, and the generally accepted point for the diastolic reading is the commencement of the fourth phase. Yet for practical purposes we often find this reading taken at the commencement of the fifth phase, that is the "silence to silence" method. Thus there is a possibility of considerable variation, especially in the diastolic figures as recorded at different institutions.

One other point of interest at this stage is that in none of the examples quoted is any mention made of the arm on which the reading is estimated. This may be quite an important point, as will be shown later.

TECHNIQUE OF ESTIMATING THE READINGS.

For the purposes of this paper the following routine was adhered to in estimating the readings. The recumbent position on the examination couch or in bed, with the arms bared and outstretched at practically the same level as the heart, was adopted. The armlet is applied as high up as possible, just below the axillary fold, with the rubber pressure bag over the brachial artery and the material of the armlet wound firmly and evenly with the tip tucked under one of the folds. The auscultatory method was used with the bell of the stethoscope placed lightly but evenly over the brachial artery just below the armlet.

The patients are warned of the sensation of tightness if it is the first occasion on which they have experienced a blood pressure estimation. A state of mental and physical relaxation is aimed at.

The bag is inflated for some little time after no sounds are heard, and the pressure is then gradually released. The systolic pressure is read at the moment when the first clicking sound comes through, that is the commencement of the first phase and the diastolic pressure at the transition from the third to the fourth phase, that is at the moment the thudding sounds change to a few dull muffled sounds which precede absolute silence.

The readings were always recorded on both arms. The instrument used was the "Tycos" sphygmomanometer (aneroid pattern), which was checked against a mercurial manometer at intervals throughout the period of this investigation.

By this routine an endeavour was made to record the pressures under approximately the same conditions for each patient on each occasion.

Material for the Investigation.

For the purposes of this work a series of individuals was observed who presented themselves for examination for some medical condition in the course of a general practice during a period of six years (1929 to 1934 inclusive). This series comprised a total of 516 patients entailing in all 6,192 examinations, or a total of 12,384 blood pressure estimations (that is both arms on each occasion).

These patients were classified into age groups as shown in Table III.

Table III.

Group.	Age in Years.	Percentage of Total.	Proportion of Sexes.	
			Male.	Female.
A	20 to 35	14	8	6
B	36 to 50	20	9	11
C	51 to 65	37	14	23
D	66 and over.	29	10	19

It is presumed that this grouping will represent the average distribution of adult patients according to age and sex met with in the course of general practice, although there will necessarily be considerable variations according to the type of practice, whether in urban or rural, industrial or residential localities.

An analysis of the blood pressure readings recorded on the first examination of each patient, and classified according to the above age grouping, is shown in Table IV. These figures refer to the average of the readings taken on the right and on the left arms respectively, and separated according to the sexes. The systolic and diastolic pressures are shown in each instance in millimetres of mercury.

Table IV.

Group.	Male.		Female.	
	Right.	Left.	Right.	Left.
A. 20 to 35 years	146/90	124/80	141/98	112/84
B. 36 to 50 years	158/94	120/78	164/104	134/92
C. 51 to 65 years	158/103	134/90	173/110	150/102
D. 66 years and over	160/102	134/90	182/109	151/99

From Table IV several points of interest become evident. Firstly there is an appreciable difference in the readings on the two arms for all the age groups. Secondly, the figures for the right arm for all ages and for both sexes are considerably higher than the normal figures of the insurance companies, whereas the figures for the left arm more closely approach these standards, except for the female groups over fifty years of age. Thirdly, the blood pressure readings increase with advancing age at a greater rate in the females than in the males.

A more detailed study of all the figures for this series of patients (that is a total of 12,384 estimations) revealed the fact that the difference between the readings on the two arms was much greater in some individuals than in others, and was usually greater the higher the systolic reading in the right arm. Very rarely did the readings in the two arms correspond exactly, and only very occasionally was the reading higher in the left arm than in the right.

In Table V these patients are classified into two groups termed respectively normal and abnormal. Normal are those in whom there is a difference of less than 20 millimetres of mercury in the systolic or less than 10 millimetres of mercury in the diastolic figures for the two arms. Abnormal are those in whom there is a difference of more than 20 millimetres of mercury in the systolic or more than 10 millimetres of mercury in the diastolic figures for the two arms. These two groups are further subdivided in accordance with the age grouping and sex.

Table V.

Group.	Age Group.	Percentage.	Male.	Female.
Normal 40%, that is small differences between the two sides.	A	5	3	2
	B	8	3	5
	C	16	7	9
	D	11	3	8
Abnormal 60%, that is large differences between the two sides.	A	8	4	4
	B	10	5	5
	C	22	8	14
	D	20	5	15

This classification of "normal" and "abnormal" is purely an arbitrary one, but, as will be shown subsequently, it appears to have some practical significance.

The average pressures obtained from all these estimations over the whole period of observation, and subdivided according to age, sex, and the degree of difference in the readings on the two arms, are shown in Table VI.

TABLE VI.

Age Group.	Male.				Female.			
	Small Differences.		Large Differences.		Small Differences.		Large Differences.	
	Right.	Left.	Right.	Left.	Right.	Left.	Right.	Left.
A. 20 to 35 years ..	150/92	134/84	143/90	116/76	130/85	112/80	149/103	116/86
B. 36 to 50 years ..	157/94	146/87	153/99	110/77	162/100	145/93	164/107	123/85
C. 51 to 65 years ..	136/93	124/88	173/111	137/87	172/110	162/106	174/108	142/90
D. 66 years and over ..	162/110	152/105	157/97	124/79	178/114	163/108	183/114	144/94

A survey was then made of all the deaths known to have occurred during this period, and these have been grouped according to whether the patients concerned exhibited large or small differences in the pressures of the two arms as recorded on their first examination. The details of these patients as to occupation, sex, age, blood pressure readings, urinary findings and cause of death have all been correlated, and are shown in tabulated form in Tables VIIA and VIIB.

Several points of interest emerge from this survey, as follows:

The sex distribution was approximately the same, each group showing a slight preponderance of females.

In the group with small differences there were only two patients of a total of twenty-one who were under sixty years of age at death, whereas in the group with large differences there were no less than twelve of a total of fifty who were under sixty years of age at death.

The urinary findings varied with the two groups. With a small difference in the two pressures it was exceptional to find any gross urinary changes, whereas with the large difference the reverse was the case, and it was unusual with these patients to find the urine normal.

The most frequent causes of death in the small difference group were malignant disease, myocarditis with cardiac failure and pulmonary tuberculosis. This is in marked contrast to the large difference group in which the outstanding causes of death were cerebral vascular lesions, *angina pectoris* or uræmia.

The number of deaths in the large difference group was just two and one-half times that in the small difference group.

Next an analysis was made of all the patients with a diastolic pressure of over 100 millimetres of mercury who have survived the six-year period of observation. These results are shown in Table VIII, and from these figures it is seen that only four out of the nineteen who have survived this period have maintained good health in addition. A glance at this table will serve to demonstrate the prognostic importance of a raised diastolic pressure.

A further group is shown in Table IX to indicate the present condition of patients who were over seventy years of age at the time of the first examina-

TABLE VIIA.

Details of Deaths during the Period of Observation among Patients with Small Differences in the Readings on the Two Arms.

Number.	Occupation.	Sex.	Age.	Blood Pressure.		Urine.	Cause of Death.
				Right Arm.	Left Arm.		
1	Home duties.	F.	61	160/110	150/100	N.A.D.	Malignant *cervix uteri*.
2	Home duties.	F.	76	160/100	162/110	N.A.D.	Myocarditis, asthenia, cardiac failure.
3	Builder.	M.	73	152/110	144/100	N.A.D.	Myocarditis, anasarca, cardiac failure.
4	Hotelkeeper.	M.	76	150/90	136/85	Albumin-trace.	*Angina pectoris*.
5	Pensioner.	F.	84	142/100	130/94	N.A.D.	Myocarditis, cardiac failure.
6	Home duties.	F.	72	158/110	140/100	Albumin-trace.	Cerebral thrombosis, cardiac failure.
7	Collector.	M.	62	110/85	100/80	Albumin-trace.	Myocarditis, cardiac failure.
8	Farmer.	M.	61	110/70	100/70	N.A.D.	Pulmonary tuberculosis.
9	Pensioner.	F.	60	120/86	110/76	N.A.D.	Malignant colon, toxæmia.
10	Manager.	M.	79	120/80	120/75	N.A.D.	Carcinoma of pancreas, asthenia.
11	Home duties.	F.	61	122/90	110/85	Albumin-trace.	Cerebral tumour (malignant).
12	Pensioner.	F.	67	110/82	100/74	N.A.D.	Carcinoma of stomach.
13	Home duties.	F.	74	104/70	100/64	N.A.D.	Malignant ovarian cyst with metastases.
14	Secretary.	F.	40	95/75	90/70	N.A.D.	Pulmonary tuberculosis.
15	Pensioner.	F.	78	145/112	132/100	Albumin-trace.	Myocarditis, cardiac failure.
16	Estate agent.	M.	70	140/85	124/76	N.A.D.	Carcinoma of the rectum, broncho-pneumonia.
17	Home duties.	F.	80	145/84	132/78	N.A.D.	Malignant colon, asthenia.
18	Pensioner.	M.	80	144/90	132/84	N.A.D.	Enlarged prostate, myocarditis.
19	Carpenter.	M.	85	116/78	104/70	N.A.D.	Malignant colon, myocarditis.
20	Home duties.	F.	35	108/82	100/72	N.A.D.	Pulmonary tuberculosis.
21	Home duties.	F.	72	115/80	100/70	N.A.D.	Carcinoma of stomach and breast.

N.A.D.—no albumin, no sugar, and microscopically clear.

TABLE VIIB.

Details of Deaths during the Period of Observation among Patients with Large Differences in the Readings on the Two Arms.

Number.	Occupation.	Sex.	Age.	Blood Pressure.		Urine.	Cause of Death.
				Right Arm.	Left Arm.		
1	Home duties.	F.	65	186/120	120/90	Albumin ++. Casts present. Sugar ++.	Hemiplegia (left), uræmia.
2	Home duties.	F.	68	235/142	155/90	Albumin +. Sugar +++.	Ataxia, hemiplegia (right), uræmia.
3	Machinist.	F.	42	286/175	200/142	Albumin ++++. Casts ++.	Chronic nephritis, anasarca, uræmia.
4	Blacksmith.	M.	64	290/180	240/180	N.A.D.	*Angina pectoris.*
5	Labourer.	M.	68	230/130	180/100	Albumin-trace.	Cerebral thrombosis, aphasia, cardiac failure.
6	Ironworker.	M.	67	190/140	164/120	N.A.D.	(Later albuminuria.) Cerebral thrombosis, uræmia, broncho-pneumonia.
7	Retired glassworker.	M.	79	180/120	155/100	Albumin-trace. Sugar ++++ (7%).	Cerebral thrombosis, broncho-pneumonia.
8	Storekeeper.	M.	63	245/170	192/132	N.A.D.	(Old tabetic.) Hemiplegia (right, then left). Intraventricular hæmorrhage.
9	Home duties.	F.	72	244/124	152/92	N.A.D.	(Survived tuberculous spine for last three years), pyobacilluria, uræmia, bronchopneumonia.
10	Retired bank inspector.	M.	71	180/120	140/98	Albumin-trace.	*Angina pectoris.*
11	Labourer.	M.	58	110/80	80/60	N.A.D.	Bradycardia, carcinoma of the stomach.
12	Blacksmith.	M.	72	244/122	192/100	Albumin-trace.	*Angina pectoris.*
13	Home duties.	F.	71	236/100	200/100	Albumin-trace. Sugar ++++ (8%)	*Angina pectoris*, acute pulmonary œdema.
14	Home duties.	F.	75	232/140	184/112	N.A.D.	Hemiplegia (right, then left), intra-ventricular hæmorrhage.
15	Home duties.	F.	69	172/110	140/100	N.A.D.	Carcinoma of the stomach.
16	Medical practitioner.	M.	54	225/172	165/120	Albumin ++. Casts +.	Cerebral hæmorrhage.
17	Printer.	M.	65	165/110	114/92	Albumin +.	Myocarditis, cardiac failure.
18	Home duties.	F.	62	230/60	166/70	Albumin ++++. Casts ++. Blood +.	Uræmia (clinically, not aortic incompetence.)
19	Ganger.	M.	54	165/100	110/80	N.A.D.	Syphilitic aortitis, aortic aneurysm, cardiac failure.
20	Pensioner.	M.	74	150/100	116/90	N.A.D.	Mental degeneration, broncho-pneumonia.
21	Home duties.	F.	66	240/132	204/116	Albumin ++.	Hemiplegia (right), broncho-pneumonia.
22	Investor.	M.	67	145/92	110/60	Albumin ++.	Severe anæmia, cardiac failure, myocarditis.
23	Manager.	M.	21	160/114	120/100	Albumin ++. Casts +.	Chronic nephritis, chronic alcoholic gastritis.
24	Home duties.	F.	66	160/100	130/92	Albumin ++.	Hemiplegia (right), intraventricular hæmorrhage.
25	Home duties.	F.	73	194/124	160/100	Albumin +.	Aortic aneurysm, cardiac failure.
26	Home duties.	F.	74	152/100	120/85	Albumin-trace.	Carcinoma of stomach.
27	Secretary.	M.	86	195/120	155/85	N.A.D.	Cerebral thrombosis, cardiac failure (three occasions).
28	Home duties.	F.	73	190/100	160/92	Albumin +. Sugar ++.	Malignant pancreas, asthenia.
29	Housekeeper.	F.	64	250/140	200/112	Albumin-trace.	*Angina pectoris.*
30	Leadworker.	M.	47	120/90	92/74	N.A.D.	Pulmonary tuberculosis.
31	Home duties.	F.	66	165/110	130/92	N.A.D.	*Angina pectoris.*
32	Pensioner.	F.	80	216/120	180/105	Sugar +++.	Diabetic gangrene of foot, toxæmia.
33	Watchman.	M.	63	165/110	120/80	Albumin +. Sugar +.	Diabetic gangrene of toes, toxæmia.
34	Home duties.	F.	74	210/140	180/125	Albumin +.	Cardiac failure, anasarca.
35	Home duties.	F.	75	145/100	120/85	Albumin +.	Bronchopneumonia, cardiac failure.
36	Gardener.	M.	70	165/110	124/82	N.A.D.	*Angina pectoris.*
37	Home duties.	F.	71	220/140	190/120	Albumin ++.	Hemiplegia (right), uræmia.
38	Home duties.	F.	46	260/170	220/145	Albumin +++.	Uræmia.
39	Pensioner.	F.	58	176/100	140/95	Albumin +.	*Angina pectoris.*
40	Clerk.	M.	72	225/170	190/148	Albumin ++.	Cerebral thrombosis, broncho-pneumonia.
41	Typist.	F.	17	150/110	122/94	Albumin +++.	Chronic nephritis, uræmia.
42	Gardener.	M.	52	226/154	162/130	Albumni ++. Sugar ++.	Cardiac failure, uræmia.
43	Pensioner.	F.	79	200/120	175/105	Albumin-trace.	Bronchopneumonia, cardiac failure.
44	Pensioner.	F.	60	140/120	112/100	Albumin +++.	Hemiplegia (right), cardiac failure (four years previously).
45	Pensioner.	F.	70	210/114	172/102	N.A.D.	*Paralysis agitans*, cardiac failure.
46	Pensioner.	F.	52	175/120	142/106	Albumin ++++.	Long-standing abdominal sinuses. Lardaceous disease, cardiac failure.
47	Pensioner.	F.	82	168/120	140/95	Albumin +. Sugar +.	Myocarditis, cardiac failure.
48	Home duties.	F.	77	200/120	175/110	Albumin ++.	Mental degeneration, uræmia.
49	Home duties.	F.	52	165/115	140/95	Albumin ++.	Arteriosclerosis, cardiac failure.
50	Pensioner.	F.	74	170/110	130/90	Albumin ++.	Myocarditis, bronchopneumonia.

N.A.D.=no albumin, no sugar, and microscopically clear.
+=Trace; ++=small amount; +++=large amount; ++++=loaded.

tion and who have survived the six years of observation. These data would appear to provide further confirmation of the advantage of a comparatively low blood pressure, and especially a diastolic pressure below 100 millimetres of mercury, as an accompaniment of advancing years, especially as judged by the criteria of longevity and good general health.

Grouping of Patients.

An investigation of this group of patients as a whole allows of their differentiation into a number of types, as under.

TABLE VIII.

Present Condition of Patients with a Diastolic Pressure of over One Hundred Millimetres of Mercury who have Survived the Six-year Period.

Urine.	Age (1934).	Sex.	Diastolic Pressure in Millimetres of Mercury on Right Arm.		Present Condition.
			1929.	1934.	
N.A.D.	72	F.	124	118	Vision greatly impaired, attacks of vertigo, invalid.
Albumin +.	65	F.	112	114	Giddiness, pensioner, dyspnœa.
Albumin ++.	76	M.	110	140	Semi-uræmic at times, invalid.
Albumin +.	71	F.	130	114	Well, fairly active.
N.A.D.	72	F.	120	110	Moderately well, but easily fatigued.
Sugar occasionally.	66	M.	104	122	Double hemiplegia, chronic invalid.
Albumin +.	68	F.	120	106	Fairly active.
Albumin +.	76	M.	160	142	Attacks of vertigo and intermittent claudication, very feeble.
Albumin +. Sugar ++.	66	F.	120	120	Complete heart block, invalid.
N.A.D.	68	F.	140	140	Tailoress, very well.
N.A.D.	46	M.	120	120	Well and very active.
N.A.D.	67	M.	110	140	Anginal attacks, semi-invalid.
Albumin ++.	68	F.	120	112	Hemiplegia three times, chronic invalid.
Albumin-trace.	68	F.	110	130	Vision impaired, anginal attacks.
N.A.D.	53	F.	130	172	Headaches, deafness, invalid.
Albumin +.	61	F.	160	170	Frequent headaches, impaired vision.
N.A.D.	38	M.	110	112	Headaches, impaired vision, invalid.
N.A.D.	76	M.	110	110	Physically active, insane.
N.A.D.	68	M.	140	160	Almost blind, anginal attacks, pensioner.

N.A.D.=no albumin, no sugar, and microscopically clear.
+=Trace; ++=small amount; +++=large amount; ++++=loaded.

TABLE IX.

Present Condition of Patients over Seventy Years of Age at the Onset who have Survived the Six-year Period.

Sex.	Age. (1934.) Years.	Blood Pressure Readings on First Examination.		Present Condition.
		Right.	Left.	
F.	88	125/80	104/70	Well and active, mentally alert.
M.	79	130/90	110/75	Very active, has glycosuria and enlarged prostate.
F.	78	140/84	132/78	Chronic arthritis of left hip, otherwise well.
F.	78	142/92	130/84	Had severe bronchopneumonia, very well now.
F.	77	110/80	95/60	Survived bronchopneumonia twice, very frail now.
M.	87	110/80	90/74	Very well physically and mentally.
F.	85	160/100	150/84	Still very active, mentally alert.
M.	77	145/100	130/100	Easily fatigued, otherwise well.
F.	77	152/100	144/94	Chronic pyobacilluria, otherwise well.
F.	77	140/90	130/90	Several attacks of biliary colic, otherwise well.
M.	78	110/80	90/60	Survived operation for fistulæ, very well.
M.	77	190/130	165/120	Had monoplegia of right arm, apparently well.
M.	78	100/80	84/62	Very well.
M.	79	140/96	140/84	Survived severe bronchopneumonia, very well.

Young Adults.

Pregnant Women.—In pregnant women the pressures are usually low and with small differences between the two sides. These patients generally progress satisfactorily.

Neurasthenics.—In neurasthenics the pressures may show one of two extremes—considerably higher than normal, or very low—but in either case with small differences between the two sides. These patients almost invariably exhibit a gross phosphaturia, and their prognosis is generally quite good. They comprise chiefly school teachers, students, and young individuals with business or domestic worries.

Toxæmic Group.—The toxæmia in this group was due to septic infections from teeth or tonsils or furunculosis, alcoholism and excessive smoking, and especially post-influenzal and tuberculous conditions were usually manifested by asthenia, giddiness and faint turns or general debility and accompanied by low pressures, sometimes with appreciable differences between the two sides. The last mentioned finding was often an accompaniment of a poor prognosis.

Hard Work or Heavy Physical Effort Group.—Hard work or physical effort was generally accompanied by a raised pressure with marked differences between the two sides, especially if the effort was constantly sustained over a long period. Should these findings be accompanied by renal changes or a family history of arteriosclerosis, the prognosis becomes considerably worse.

Middle-Aged Patients.

Males.—Business or domestic worries were frequently found, accompanied by raised pressures with marked differences on the two sides, and a condition of glycosuria which is not diabetic in character. Arteriosclerotics with a certain degree of cardiac involvement often showed high pressures

with only slight differences between the two sides and no renal pathology. In pernicious anæmia the pressures were always low and almost equal on the two sides; these always improved when the blood picture approached the normal under liver treatment. A number of males exhibited some degree of endocrine dysfunction accompanied by nervous symptoms, tachycardia, giddiness, and raised pressures with little variation between the two sides. These symptoms were somewhat akin to climacteric changes in the female.

Females.—In women about the climacteric period there were frequently seen elevated pressures which were very unstable and which varied greatly on the two sides. This condition often persisted for two years or more and then gradually approached the normal. Worry or mental stress of any kind will always accentuate the condition in these patients.

Both Sexes.—In both sexes hemiplegia often showed considerable differences in the readings on the two sides, especially at or shortly after the onset of the lesion. The pressures are generally considerably lower in the affected limb.

Hyperpietics, almost without exception, showed large differences between the two sides, and the course of the illness was usually more severe than when the pressures were more nearly equal. Should urinary changes occur in addition, the prognosis is materially worse.

With aortic incompetence there was a high systolic and low diastolic figure and marked variations on the two sides.

In the few instances in which aneurysm was diagnosed clinically and confirmed by X ray or autopsy findings, there was not the pronounced difference usually described with the condition.

Low pressures with appreciable differences on the two sides and a slow pulse rate were on a number of occasions the accompaniments of chronic peptic ulcer or some malignant condition.

A few patients with pseudo-angina precipitated by worry or mental stress showed pressures within normal limits and only slight variations between the two arms. These all progressed very satisfactorily.

Elderly Patients.

In the elderly patient group the combination of high pressures with marked variations on the two arms almost always was of serious import, more especially if associated with albuminuria or glycosuria. On the other hand, the senile patients with normal or low pressures with small variations on the two sides seemed to enjoy good health and to lead an active existence for their years, and frequently survived severe intercurrent illnesses. Several of these people with a condition of heart block and low pressures almost equal in the two arms have carried on comfortably for a number of years.

Children.

A large number of children up to the age of fourteen years, and attending an out-patient medical clinic at the Children's Hospital for a wide variety of conditions of ill-health, showed little variation between the readings on the two sides.

Variations of Blood Pressure Readings in the Two Arms.

Regarding the question of variation of the blood pressure readings in the two arms, very little comment is made in any of the recognized text books or systems of medicine, except a passing mention that in cases of aortic aneurysm the pressures may be unequal in the two arms.

A search of all the available literature also reveals comparatively few articles upon this aspect of the subject. They have been reviewed as follows.

Riesman[1] states that it is advisable to take the pressure in both arms; not infrequently it varies on account of sclerotic processes, cervical rib, or aneurysm. The blood pressure on the weaker side would then give a false impression.

Kay and Gardner[2] published the results of 500 readings on 125 patients. They regarded variations of five to ten millimetres of mercury in the systolic or diastolic readings of the two sides as normal. With this as a criterion, they found 100 patients (or 80%) within normal limits and 25 (or 20%) as distinctly abnormal. Arteriosclerotic and hypertensive patients more often show these variations. The greatest differences were found in those with *angina pectoris* or aortitis. The group which is second in importance are those patients who have vaso-motor disturbances, such as occur at the menopause. Patients above forty-five years have the most marked variations. Sex apparently plays no part. The right arm is the highest in the majority of cases. They believe that the cause of these differences is varying functional changes occurring in the blood vessels of the two arms.

Hurwitz[3] quoted a patient, aged fifty years, with a systolic pressure of 250 millimetres of mercury (right) and 150 millimetres of mercury (left), which readings remained fairly constant. The other clinical and X ray findings were normal. He asked for a possible explanation. The editorial reply states that frequently a satisfactory explanation cannot be found for cases exhibiting such a difference in pressure. A double aorta may cause such a picture, the left subclavian coming off the anterior and smaller arch, just before the two reunite above the *ductus arteriosus*. The œsophagus lies between the two, and barium will show it in front of the larger arch. The condition may not show in a plate, or fluoroscopically, without barium. It is a rare condition.

The hypoplasia of the aorta involving the arch is a possibility. Whether such a picture is congenital or acquired is not certain.

Plaques of the aorta will have been mentioned as a cause, especially when they are associated with proliferative changes. An aneurysm of the arch may escape detection with the most careful X ray examination. One such case was due to a dissecting aneurysm showing no enlargement on X ray examination and found only at necropsy.

Korns and Guinand[4] also report on the bilateral brachial pressure measurements in 1,000 normal subjects. They stated that significant sphygmic inequality occurred 439 times in 378 people. Significant inequalities in pulse pressures appeared in 274 persons, 67 of whom failed to show differences

of 16 millimetres of mercury or more between the two systolic or diastolic levels. Nearly three-fourths of the higher pulse pressures were dextro-lateral. These pressures were measured simultaneously in the two arms, but for all practical purposes consecutive measurement gives equally satisfactory results.

Sphygmic inequality without organic disease is probably always transitory, and it is reasonably certain that all normal persons manifest it at one time or another.

The inequality may involve only the systolic pressures, or only the diastolic, or both, and if the two levels are disparate the inequality may be concordant (that is both right pressures higher than both left, or *vice versa*) or discordant (that is the right systolic pressure higher than the left and left diastolic higher than the right, or *vice versa*).

In some persons the higher pressure is irregularly heterolateral, in others it appears to be always homolateral.

There is no evidence that right handedness or left handedness plays any part. The physiology of transitory brachial disparities is not understood. Sphygmic inequality in brachial or carotid arteries cannot be regarded as a sign of disease of the aorta or its branches, unless it can be shown to be permanent.

Norris[5] mentions that bilateral inequality of pressure is frequent in arteriosclerosis.

MacLaren[6] observed that normally there may be a difference in the systolic pressure of the two arms up to 10 millimetres of mercury. In only 12% is the difference as great as 20 millimetres of mercury. Therefore an increased difference calls for some explanation, which is probably in some abnormal condition of the aortic arch.

It is obvious that each of these authorities, whilst recognizing the variation of the readings in the two arms, regards them as either of slight degree or of comparative rarity. As to explanation of the disparity, this has simply been a matter of conjecture, and some of the conditions suggested as causative factors are certainly of much rarer occurrence than is the difference of pressures even in the figures quoted by these observers, for example cervical rib, aneurysm, functional changes in the blood vessels of the two arms, double aorta or hypoplasia of the aortic arch.

As the series of patients at present under review showed the variation in the readings of the two sides to be often of considerable degree and of very frequent occurrence it seemed to me that there is possibly a simpler and more reasonable explanation of this clinical observation, and this is now submitted for discussion.

Suggested Explanation of the Variation in Blood Pressure Readings in the Two Arms.

Let us consider first a few elementary mechanical or hydrostatic principles which might be applied in solving this problem.

In Figure II there is represented a straight rigid tube through which a liquid medium is flowing in a steady stream in the direction from A to B. It is an axiom of hydrostatics that the pressure of the stream at B must be less than the pressure at A, otherwise the liquid would not flow in this direction. In view of this fact it will be evident that in the three small outlets all of equal bore and at right angles to the main tube the pressure in X will be greater than that in Y, which in turn will be greater than that in Z.

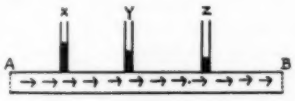

Figure II.

In Figure III the same system has been converted into an evenly curved tube. In this system there will be a further dissipation of energy, (*a*), as the direction of the stream is changed from point to point in moving from A^1 to B^1 and, (*b*), as there will be greater friction from the walls in the curved tube than in the straight one.

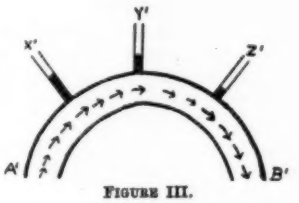

Figure III.

This simply means that there will be a still greater drop in pressure as the stream moves from X^1 to Y^1 to Z^1 in this system in comparison to the straight tube.

In Figure IV a system is represented in which the curve of the main tube is much more pronounced and in which the three outlets are closely approximated in one portion of the tube; but instead of being taken off at the right angles from the main tube, the direction of each outlet varies, so that X^2 is in a direction against the flow in the main stream, Y^2 is at right angles to the main tube, and Z^2 is more closely in the direction of the main stream.

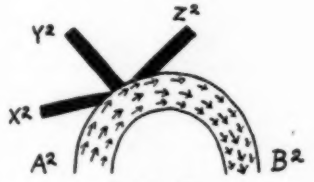

Figure IV.

In this instance the pressure in X^2 would be expected to be less than that in Y^2, which in turn should be less than that in Z^2. These differences should be considerably more pronounced if the stream of fluid is being forcibly pumped against some resistance rather than flowing steadily and continuously, and if the main tube is of an elastic, not a rigid material.

With the object of confirming or disproving this supposition a system was constructed from a piece of large bore rubber tubing with outlets of finer

tubing corresponding to the plan of Figure IV. The smaller outlet tubes were connected with the main tube in such a way that their communicating ends were quite flush with the inner surface of the large tube without any projecting portion within the lumen which would in any way interrupt the stream.

Each outlet tube was connected with a small mercury manometer in order to register the actual pressure in each individual tube. The whole apparatus was mounted on a board for stability.

The main tube was connected with a hose attached to the ordinary household water supply, which thus provided the moving fluid stream.

The experiment was carried out in three sections:

Section I.—A continuous stream of water was allowed to flow uninterruptedly through the system and the readings in the manometers were observed at varying speeds of the stream regulated by the degree to which the tap was turned on. Each performance was repeated on ten occasions and the average reading was calculated.

Section II.—With still a continuous stream of water these experiments were repeated, but this time with the outlet from the main tube considerably narrowed in order to create what would correspond to an increased peripheral resistance. With this modification each performance was repeated on ten occasions, as before, and the average readings again calculated.

Section III.—The same proceeding was then repeated with a much narrower outlet from the main tube, that is, a considerably increased peripheral resistance, and in addition, an intermittent stream regulated by rapidly turning the tap from the water supply alternately on and off. (This technique was found to be preferable to the use of a Higginson pattern syringe connected up to the apparatus and the force provided by alternately squeezing and relaxing the bulb.)

This proceeding was repeated thirty times, and the average reading was calculated for each manometer.

The figures for these sections of the experiment are shown in Table X.

Table X.

Showing Pressures in Millimetres of Mercury Recorded by the Manometers at X_2, Y_2, and Z_2.

Section.	X_2.	Y_2.	Z_2.
I	23	34	47
II	99	117	146
III	163	182	217

The results of these experiments would therefore seem to indicate that the pressure in an outlet tube from a large curved main supply tube, with the contained stream being forced along intermittently, will be greater if the direction of that outlet tube is in close alignment with the direction of the flow than it would be from a similar bore outlet tube from an adjacent part of the main supply, but with its line of direction either away from or at right angles to the main curved tube.

Let us now direct our attention to Figure V, which is a diagrammatic representation of the relative positions of the main branches from the arch of the aorta.

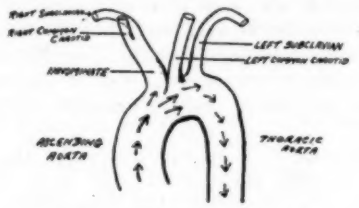

Figure V.
Diagram of the aorta and its branches from the left and in front. (*After* Spalteholtz.)

The innominate artery, from which arises the right subclavian, is more in the direct line of the stream of blood in its upward thrust from the ascending aorta, whereas the left subclavian arises further along the arch of the aorta and in a direction almost at right angles to the flow.

From this anatomical arrangement of the great vessels, and in the light of the experimental work just described, it would appear reasonable to assume that the pressure in the right subclavian (and consequently in the axillary and brachial arteries) would, in ordinary circumstances, be greater than that in the left subclavian (and consequently in the axillary and brachial arteries of that side).

This is offered as a simple and rational explanation of the difference in the blood pressure readings estimated at the brachial artery in the two arms which has been shown to be of frequent occurrence during the clinical examination of the patients now under review.

A series of twelve charts is shown herewith as representing the various clinical types encountered and demonstrating numerous outstanding features discussed in the course of the paper. These charts will be found to be self-explanatory and do not require a detailed description of each.

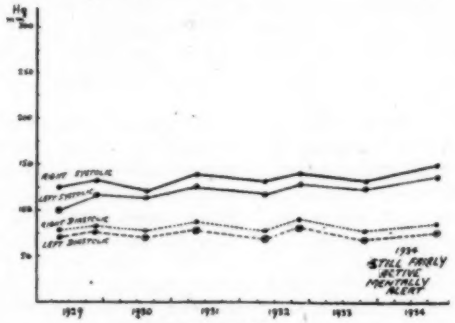

Figure VI.
Case 15.—A female, aged eighty-two years, with senility, asthenia and pyobacilluria.

Summary.

The results published in this paper are the outcome of some observations made during a period of six years in a general practice. These comprise a total of 12,384 blood pressure readings (that is 6,192 examinations) on a group of 516 patients.

The risks associated with a systolic pressure persistently raised above the apparently severe standards of normality demanded by most life insurance offices have been fully demonstrated.

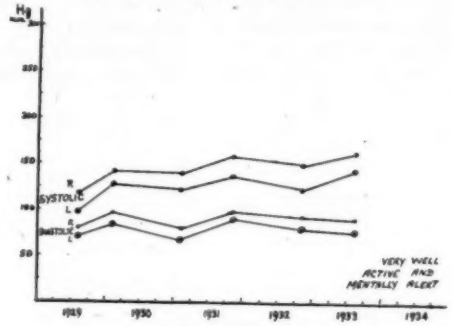

FIGURE VII.

CASE 321.—A male, aged eighty-three years, with prostatic enlargement and cystitis.

The significance of a raised diastolic pressure (above 100 millimetres of mercury) and its importance as a prognostic aid are particularly emphasized.

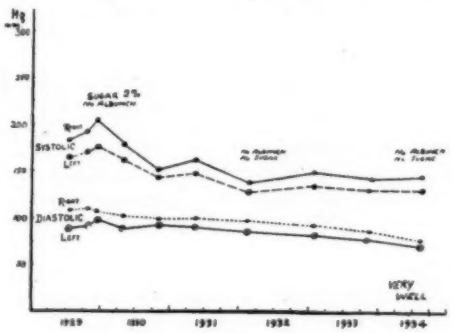

FIGURE VIII.

CASE 273.—A female, aged forty-six years, at the menopause, nervy and suffering from glycosuria.

The frequent occurrence of differences in the readings on the two arms (often appreciable in degree) is noted and an explanation is offered for the difference.

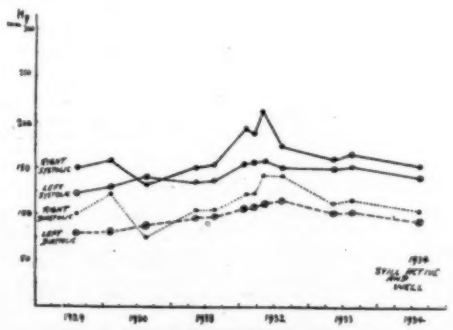

FIGURE IX.

CASE 476.—A female, aged seventy-three years, with senile myocarditis and osteoarthritis.

In any clinical examination of an individual the blood pressure readings should be taken on both arms and considered in conjunction with the urinary findings before the state of the cardiovascular system is assessed.

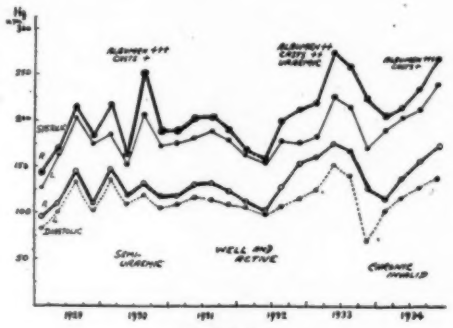

FIGURE X.

CASE 19.—A male, aged seventy-two years, with arteriosclerosis and chronic nephritis.

The practical application of these findings assumes considerable importance from various aspects, for example, (i) in medical practice from the point of view of diagnosis, prognosis and treatment; (ii)

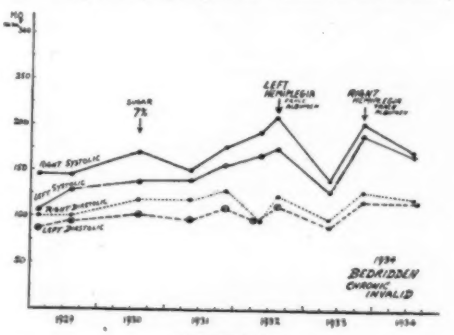

FIGURE XI.

CASE 31.—A male, aged sixty-four years, who was diabetic and arteriosclerotic.

in life insurance examinations for a correct estimation of the expectation of life for any individual; (iii) in various government and departmental and industrial medical examinations for the determination

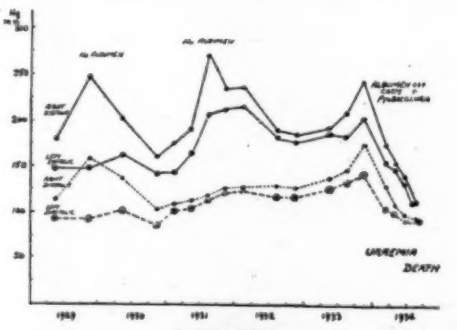

FIGURE XII.

CASE 47.—A female, aged seventy-one years, who had old tuberculosis of the spine and arteriosclerosis.

of physical fitness or incapacity for various occupations or for the granting of pensions.

The necessity for a uniform technique in the estimation of blood pressure readings and for uniform

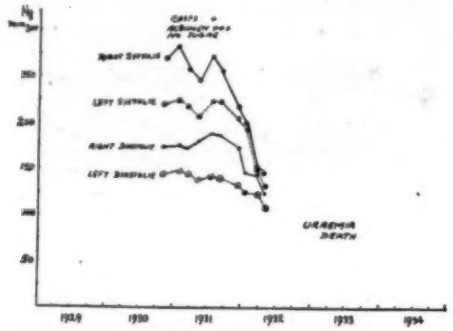

FIGURE XIII.

CASE 141.—A female, aged forty-one years, with chronic nephritis and hyperpiesia.

criteria as to the point for recording the systolic and diastolic figures is particularly stressed.

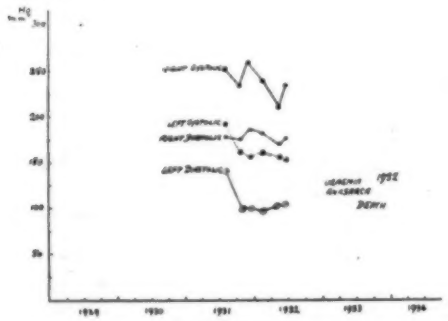

FIGURE XIV.

CASE 423.—A male of fifty-two years, with arteriosclerosis, diabetes and cardiac failure.

Finally, in this connexion, it is suggested that a standard of normal blood pressure readings for healthy

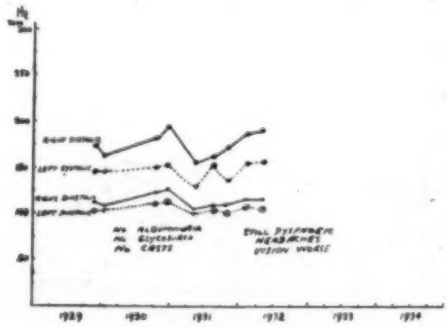

FIGURE XV.

CASE 197.—A male, aged thirty-two years, suffering from dyspnœa, headache and defective vision.

persons in Australia might be produced by coordination of the life insurance companies of the various States. Such a table would be extremely valuable for practitioners throughout the Commonwealth.

ACKNOWLEDGEMENTS.

In conclusion I should like to record my deep appreciation of the assistance so generously afforded me by the managers, medical officers, and officials

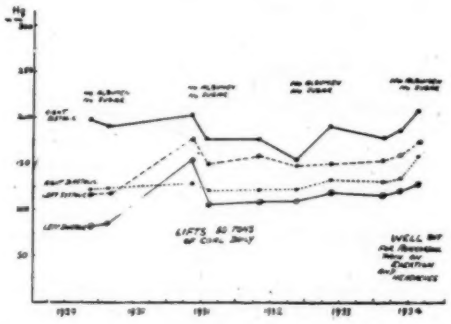

FIGURE XVI.

CASE 69.—A male of forty years, with arteriosclerosis.

of all the life insurance companies to which I appealed for help in connexion with this work; of the helpful discussions with my partner, Dr. Carl V. Stephens, from which originated this piece of clinical research; and of the valuable support of my wife in helping to carry out the details of the experimental work.

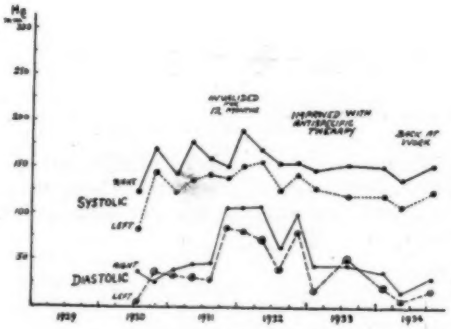

FIGURE XVII.

CASE 376.—A male of sixty years, with aortic regurgitation, multiple arthritis, and reacting to the Wassermann test.

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TORULOSIS, WITH A REPORT OF A CASE OF MENINGITIS DUE TO TORULA HISTOLYTICA.

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The main purpose of this paper is to report a case of meningitis caused by the organism known as *Torula histolytica*. Although comparatively few cases of infection due to *Torula histolytica* have been reported in the literature of the world and especially in the literature of Australia, we feel that many more cases of this type of infection may have occurred and will occur. With this fact in mind we have endeavoured to give a brief review of the literature on the subject, with a fairly full account of the disease from the clinical, pathological and bacteriological aspects. A detailed account of the methods we employed to isolate and identify the organism from the case described is given, together with the results of our animal experiments.

Definition.

Torulosis is a disease caused by an organism known as *Torula histolytica*. This organism is a species of yeast-like fungus which reproduces by budding alone and does not produce endospores or a mycelium. It is also said not to ferment sugars.[1] The parasite invades both the animal and human body.

A careful study of the cases reported in the literature shows that the parasite has a special affinity for the central nervous system, and in practically all cases this system was affected. The pulmonary system is the next most often affected and very often these two systems are affected together. Henrici[2] states that the primary lesion occurs in the lung. Other organs are known to have been involved and a few cases of generalized torulosis have been described.

Pathologically the disease resembles the infectious granulomata, and it is often difficult to distinguish it from a tuberculous infection. It is stated that it may be almost impossible to distinguish the disease from tuberculosis unless the organism is found.

Historical.

In May, 1894, Gilchrist exhibited and described before the American Dermatological Association at Washington microscopic sections from a case which had been diagnosed as typical chronic scrofuloderma of the back of the hand.[3] In the tissues were found large numbers of bodies which were round, doubly contoured and refractile, and which appeared more distinct after the tissue had been treated with *liquor potassæ*. Gilchrist expressed the view that these organisms should be classed as belonging to plant life rather than to animal life. This was probably the first mention of a yeast-like infection in man.

In November, 1894, Busse[4][5][6] was responsible for the first published report of a case of yeast-like infection. He reported a case to which he gave the title *"Saccharomycosis hominis"*. The patient was a woman, aged thirty-one years, and the lesion was a localized subperiosteal inflammation of the left tibia.

In 1895-1896 much work was done on yeast-like infection in man, the principal investigators being Sanfelice[7][8][9][10][11][12] in Italy, Rabinowitsch,[13] Maffucci and Sirleo,[14][15] Fermi and Aruch,[16] Tokishige,[17] Corselli and Frisco,[18] Charrin and Ostrowsky, and Roncali.[19]

In July, 1896, Gilchrist and Stokes[20] published the report of a case showing the presence of an oidium in the tissues of a case of *pseudo-lupus vulgaris*.

In August, 1896, Curtis[21] published an excellent review of the literature with regard to yeast-like infection in man. He described a case of parasitic angina clinically resembling thrush, the infecting agent being classified amongst the yeasts. A very detailed account of his methods of investigation was also given. Curtis called his organism *Saccharomyces subcutaneus tumefaciens*.

The literature on yeast-like organisms in man then began to accumulate more rapidly, the principal contributions being in 1898 by Gilchrist and Stokes,[22] and by Wells, in 1899 by Hektoen,[23] by Hyde, Hektoen and Bevan, and by Owens, Eisendrath and Ready,[24] in 1900 by Coates and by Ophüls, and in 1901 by Ricketts[25] and by Klein.[26]

In 1902 Frothingham[27] published the first instance of torulosis as a clinical entity. He examined a tumour-like mass from the lung of a horse and isolated a torula from the lesion.

Further instances were reported each year—von Hansemann[28] (1906), Turck (1907), Brewer and Wood[29] (1908), and Rusk[30] (1911-1912), until in 1916 Stoddard and Cutler[31] published their review of yeast infections in man, which today is still a classic on the subject of torulosis. They made the first real attempt to correlate the clinical and pathological findings with the morphological and cultural characteristics of the organism. They collected four cases from the literature and added two of their own, making a total of six. Their excellent review stimulated observers to watch more carefully for cases of torulosis, and from 1916 to the present time many more cases have been reported. We have made a careful search of the literature available to us and as indexed in the "Quarterly Cumulative Index Medicus", and have found that to date (December, 1934) forty-nine cases of torulosis, either generalized or local, have been reported. The majority of these have been reported from America, while the others are from England, Germany, Australia, Japan, France, Dutch East Indies, and Italy.

A more detailed analysis of these cases will be given later.

Classification.

The classification of the yeast-like fungi has always presented considerable difficulty. A mass of literature has accumulated with reference to lesions in human beings produced by yeast-like organisms—organisms which budded without producing mycelium and those which did not bud in the tissues—all of these organisms being loosely classified under the term blastomycosis.

For many years this term blastomycosis was used to include diseases caused by *Torula*, *Oidium* and *Monilia*, and these organisms were classified as *fungi imperfecti*. These organisms include such as *Monilia psilosis*, *Oidium*, which was identified by Gilchrist, *Oidium albicans*, which is found in the condition known as thrush, and other varieties of *Oidia* and *Torula*.

Originally the yeasts were divided into two subdivisions: those forming endogenous spores and those forming no spores. The spores were regarded as ascospores, and the two groups were regarded as subdivisions of the Ascomycetes and *fungi imperfecti* respectively. Recently, however, investigation shows that the classification of the yeasts may not be so simple as was at first thought. Species have been found which seem to form exogenous spores that have been regarded as basidiospores and as conidia respectively.

If we disregard these unusual species we may classify the yeasts reasonably well into: (i) Saccharomycetaceæ, forming endogenous spores; (ii) Torulaceæ, forming no spores.

The yeasts are also grouped with the Ascomycetes, as the spores are regarded as ascospores—in some cases they are eight in number and in other cases result from a conjugation of two cells. Transitional forms between one-celled yeasts and the complex filamentous forms are also found; these transitional forms are included under the Endomyces.

The relationship of the two groups of yeasts and of transitional fungi are shown diagrammatically in Figure I, which is taken from Henrici's text book.[32]

Hansen was the first to distinguish between spore-forming and non-spore-forming yeasts, and he named the latter group Torula. Many attempts to classify this group suitably have been made, and these are adequately tabulated by Henrici. The first was that of Will (1916), who divided the Torulaceæ into: (i) *Eutorula* (fat droplets develop in this species), (ii) *Torula*, (iii) *Mycotorula* (with a rudimentary mycelium).

This was followed by other classifications, notably those of Guilliermond, Ciferri and Redaelli and Harrison.

Harrison's classification is that usually adopted. He retains the genus *Mycotorula* of Will and classifies the remaining species according to pigment production: (i) rhodotorula, red pigment; (ii) chromotorula, other than red pigment; (iii) torula, no pigment.

He further subdivides torula and mycotorula according to their sugar fermentations, classifying them into the following groups:

Group A, producing no acid or gas in any sugar.

Group B, producing slight acid in dextrose, mannose, fructose or galactose.

Group C, producing slight acid with or without trace of gas in dextrose, mannose, fructose or galactose and saccharose.

Group D, producing marked acidity and gas in dextrose, mannose, fructose or galactose.

Group E, producing marked acidity and gas in dextrose, mannose, fructose, galactose and saccharose.

Group F, producing marked acidity and gas in dextrose, mannose, fructose, galactose, saccharose and raffinose.

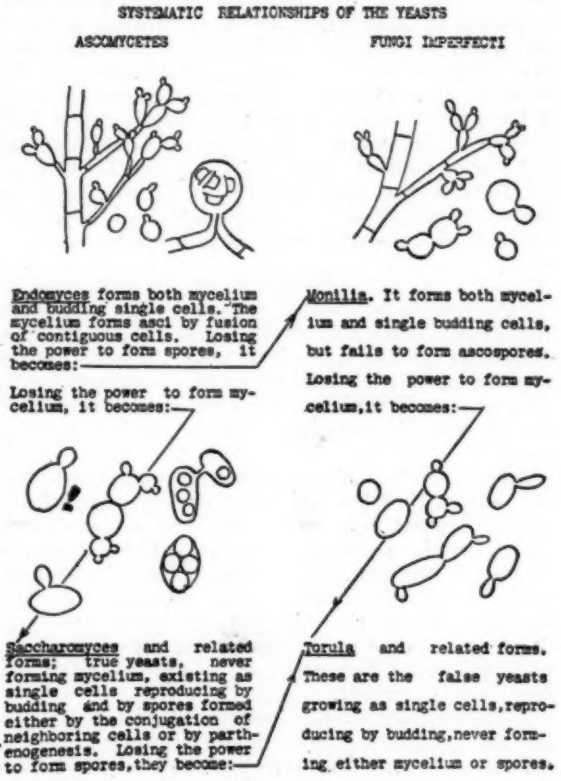

Figure I.

Group G, producing marked acidity and gas in dextrose, mannose, fructose, galactose, saccharose and maltose.

Group H, producing marked acidity and gas in dextrose, mannose, fructose, galactose, saccharose and lactose.

Group I, producing marked acidity and gas in dextrose, mannose, fructose, galactose, saccharose, lactose and inulin.

It remained for Stoddard and Cutler[31] to produce order out of chaos and to differentiate the different diseases which had been grouped under the term blastomycosis. They classified the infections

as: (i) true yeast infections, (ii) torula infections, (iii) oidiomycosis, (iv) coccidioidal granuloma.

For the differential points of these infections reference may be made to Table I, which is taken from Jacobson's text book.[33]

From its curious histolytic action, which is so characteristic of the disease, Stoddard and Cutler name the organism *Torula histolytica*.

Bacteriology.

Distribution.—Members of the genus *Torula* are found widely distributed in nature. For example, they have been isolated from wasps' and bees' nests, from the stems of plants, from grasses, and from the bodies of insects. Rogers claims to have demonstrated them in canned butter, and Klein to have found them in canned milk.

Morphology.—The organism is an ovoid or spherical cell varying from 3μ to 15μ in diameter. The organism has a definite cell wall. The cytoplasm stains poorly. In tissues the organism may be seen either in an intracellular or extracellular position, and is usually surrounded by a vacuolated space. Budding may be seen. In fresh material, such as cerebro-spinal fluid or sputum, the organisms may easily be mistaken for lymphocytes.

Reproduction.—Reproduction is by budding only. There is no mycelium or spore formation.

Staining Reaction.—The organism is Gram-positive, but in old cultures may become Gram-amphophilic.

Cultural Reactions.—The organism is an aerobe and grows quite well at room temperature and with a wide range of pH values. The most suitable medium is Sabouraud's medium with carbohydrate enrichment. The addition of 0·3% lactic acid to the medium is helpful, as it inhibits the growth of bacteria without having any effect on the torula. The organism also grows well on ordinary media.

The colonies are pinhead in size at first and appear as opaque, yellowish-white plaques. They are raised, circular, smooth and moist. Later they become confluent, and old cultures have a yellowish colour.

Biochemical Reaction.—It is definitely stated that *Torula histolytica* does not ferment carbohydrates. The use of the term ferment in this statement must mean only that no gas is produced, as most workers seem to agree that acid is formed in some of the carbohydrates. Frothingham[27] states that no fermentation occurred after ten weeks. Stoddard and Cutler[31] and Henrici[32] state that no gas was produced, but do not mention if there was acid formation. Sheppe,[34] Stone and Sturdivant,[35] Watts,[36] and Fitchett and Weidman[37] report the formation of acid without gas production in various carbohydrates. Weidman[38] says that the capacity of *Torula histolytica* for fermenting carbohydrates and other substances is almost nil; of nineteen strains from cerebro-spinal cases, all produced acid in glucose and lævulose; gas was not produced. He found great irregularity in the reactions, and some strains produced acid in glucose, lævulose and saccharose, as well as maltose or raffinose or inulin or mannite. Rappaport and Kaplan[39] state that no change was produced with the common carbohydrates. We have found delayed acid formation without gas production in some carbohydrates, and the details of our findings will be given later.

Animal Inoculation.—The organism is pathogenic for white mice, rats and, to a lesser degree, rabbits.

Table I.

Fungus Diseases. Differential Features of Yeasts, Torula, Oidiomycosis, and Coccidioidal Granuloma. (After Jacobson.)

	Genus.	Reproduction.	Sporulation.	Mycelium.	Sugars.	Pathogenicity.	Lesions.	Pathological Characteristics.	Size of Organisms. (Microns.)
Blastomycetes.	Saccharomycetes (true yeasts)	Buds.	Ascospores present.	Absent.	Fermented.	Feeble.	Skin.	Necrosis; epithelial overgrowth; giant cell abscess formation; polymorphonucleosis.	
	Torula ..	Buds.	Absent.	Absent.	Usually not fermented.	Moderate.	Nervous system; other organs to less extent; skin never.	Chronic inflammation, caseation occasional; no polymorphonucleosis; gelatinous matrix.	1–13
	Monilia ..	Buds.	Absent.		Fermented.	Moderate.	Mucous membranes; mouth and gastro-intestinal tract; slight skin pigmentation.	Chronic ulceration.	5–10
Fungi Imperfecti.	Oidiomycosis ..	Buds.	Absent.	Present.	Not fermented.	Slight.	Skin always; often bones, internal organs and brain.	Necrosis; epithelial overgrowth; miliary abscesses; tubercle-like nodules; polymorphonucleosis; no gelatinous matrix.	3–30
	Coccidioidal granuloma	Never buds.	Endospores present.	Present with aerial hyphæ.	Not fermented.	Marked.	All organs; often skin.	Nodules and cysts; giant cells; caseation; abscess formation; polymorphonucleosis; no gelatinous matrix.	5–85

It is also pathogenic to a limited extent for monkeys, as we shall show later. Guinea-pigs and dogs appear to be immune. The lesions produced in animals resemble those seen in man.

Clinical Manifestations.

The clinical manifestations of torulosis are many and varied, and no typical clinical picture can be described. The organism appears to have a peculiar affinity for the central nervous system and the pulmonary system, and in only about 20% of cases have other portions of the body been affected. The disease may be of two types, local and systemic torulosis.

Local torulosis is rare. Here the organism affects one particular part of the body and remains localized to that part. Cases of torulosis of the naso-pharynx, tongue, skin, spine and pelvis have been reported. The symptoms vary with the area affected and correspond to those produced by pathological lesions in these areas.

In systemic torulosis, as already noted, the central nervous system and the pulmonary system are the most commonly affected. Other organs which have been affected are the spleen, liver, kidney, mesenteric glands and ileum, in that order of frequency. The clinical manifestations vary greatly and depend not only on the site of infection, but also probably on the virulence of the organism and the natural immunity of the patient. The disease usually runs a subacute or chronic course, but acute cases have been described. The general symptoms are those of a mild or moderately severe toxæmia. There is a rise in temperature and also an increased pulse rate. When the central nervous system is involved there is usually headache of a moderately severe type. Visual disturbances, such as failing vision, diplopia and nystagmus, may be seen. Symptoms of a more chronic type of meningitis may also be evident. Mental symptoms may be present, but are rare, although some patients are recorded as having developed maniacal tendencies.

When the pulmonary system is involved, the signs and symptoms are similar to those seen in other infections of the lung and may easily be mistaken for tuberculosis, syphilis or other types of pulmonary mycosis. Great variations in the signs and symptoms are met with. As pulmonary torulosis is usually associated with torulosis of the central nervous system, both sets of symptoms may be seen.

Treatment.

The localized type of the disease may be treated by radical surgical methods with complete resection of all accessible lesions. It is probably best to use the actual cautery knife.

The systemic type of the disease does not appear to yield to treatment. The use of colloidal copper and a torula vaccine has been suggested, but, so far as we know, this has not yet been tried.

Pathology.

Infection by *Torula histolytica* produces in the tissues a defensive cellular reaction which resembles that of an infectious granuloma, except in the brain, where the lesions are somewhat different in character.

In the lungs and other organs the main macroscopic appearance is that of fine pinpoint nodules resembling the tubercles seen in a miliary tuberculosis. These tubercles vary in size from about 0·5 to 8·0 millimetres in diameter.

Microscopically the structure of these tubercles resembles very closely that of the tuberculous follicle. There is a definite concentric arrangement of endothelial cells and lymphocytic cells. Giant cells are usually present within the follicle. One is struck by the almost entire absence of polymorphonuclear cells. The organisms may be detected within the follicle.

In the central nervous system the reaction of the tissues to the organism is somewhat different. The resultant lesion is really a chronic leptomeningitis. The meninges become considerably thickened and adherent, and scattered over the surface can be seen multiple pinpoint nodules similar to the tubercles seen in a tuberculous meningitis. There is very little exudate. In the brain there may be either the formation of a perivascular granuloma or nodules or cyst-like spaces scattered throughout the brain substance, which assumes a gelatinous appearance.

The microscopical picture of the lesions is rather striking. Sections through the nodules on the meninges show a structure similar to that of tuberculous follicle, with an almost entire absence of polymorphonuclear cells. Giant cells are usually present. The torulæ may be present in these nodules.

Sections through the lesions within the brain substance show an actual solution of the brain substance, and it is this histolytic action of the organism which has given it its name. The organisms can be seen lying in clear cyst-like spaces. Around these spaces there is very little cellular reaction, although mononuclear cells and lymphocytes may be present.

The formation of these gelatinous spaces has formed the subject for much discussion and various theories are put forward. Stoddard and Cutler consider that they are produced by a lytic property of the organism itself. Sheppe considers that they are due to a complicated metabolic process of the living organism, while Freeman and Weidman consider that they are mechanical in origin.

Review of the Literature.

Table II, which is based on the table of cases given by Ball,[40] gives a summary of the forty-nine cases reported up till December, 1934. In forty-three of these the central nervous system was involved, and there was usually generalized torulosis. Of the other six three were definitely localized to one particular area of the body. Brewer and Wood's case[20] was localized in the vertebral muscles, McGehee and Michelson's case[41] in the inguinal region, with abscess formation (this

Table II

Number.	Observer.	Year.	Sex.	Age.	Extraction.	Lesion.
1	von Hansemann.	1906	M.	18	German.	Central nervous system.
2	Turck.	1907	F.	43	German.	Central nervous system; œsophagus.
3	Brewer and Wood.	1908	M.	20	Russian.	Vertebral muscles.
4	Rusk.	1911	F.	63	German.	Central nervous system; left lung.
5	Rusk.	1912	M.	57	German.	Central nervous system; right lung; kidney.
6	Verse.	1914				Central nervous system; skin; generalized.
7	Stoddard and Cutler.	1916	F.	42	American.	Central nervous system.
8	Stoddard and Cutler.	1916	M.	39	American.	Central nervous system; lungs.
9	Gotto.	1916	M.	61	Japanese.	Central nervous system.
10	Swift and Bull.	1917	M.	53	Chinese.	Central nervous system.
11	Pierson.	1917	M.	57	American.	Central nervous system; left lung.
12	Evans.	1922	M.	13	Mexican.	Central nervous system.
13	Evans.	1922	F.	20	Mexican.	Central nervous system.
14	Badham.	1922			Australian.	Central nervous system.
15	Barlow.	1922		5	Australian.	Central nervous system.
16	Williams.	1922	M.	35	Australian.	Central nervous system.
17	Freeman and Weidman	1923	M.	39	American.	Central nervous system; mesenteric glands.
18	Sheppe.	1924	M.	48	American.	Lung.
19	Bettin.	1924	F.	40	American.	Central nervous system; right lung.
20	Hansmann.[45]	1924	M.	45		Central nervous system.
21	Neal and Shapiro.	1925	M.	16	American.	Central nervous system.
22	Wilhelmj.	1925	M.	48		Central nervous system.
23	Lynch and Rose.	1926	M.	46	Jewish.	Central nervous system.
24	McGehee and Michelson.	1926	F.	26	Negress.	Abscess, inguinal region.
25	Rappaport and Kaplan.	1926	M.	54	Kurd.	Skin; central nervous system; lungs; kidneys.
26	McKendree and Cornwall.	1926	F.	50	American.	Central nervous system.
27	Berghausen.	1927	M.	28	White.	Tongue; unproved lung lesion.
28	Jones.	1927	M.	34	White.	Pharynx.
29	Hall, Hirsch and Mock.	1928	M.	53	American.	Central nervous system; spleen.
30	Wortis and Wightman.	1928	M.	20	American.	Central nervous system.
31	Hirsch and Coleman.	1929	F.	30	Negress.	Central nervous system; lungs, miliary.
32	Maner by Ball.	1929	M.	54	American.	Central nervous system.
33	Ball.	1929	F.	50	English.	Central nervous system; lungs, ileum.
34	Stone and Sturdivant.	1929	M.	50	American.	Central nervous system; lungs.
35	Sheppe.	1929	M.	42	American.	Central nervous system.
36	Semerak.	1929				Central nervous system.
37	White.	1930				Central nervous system.
38	Smith and Crawford.	1930	F.	31	English.	Central nervous system.
39	Massee and Rooney.[46]	1930	F.	32	American.	Central nervous system.
40	Urbach and Zach.	1930	M.	27		Skin; central nervous system; mouth; generalized.
41	Reale.	1931				Central nervous system.
42	Freeman.	1931				Central nervous system.
43	Watts.	1932	F.	32	American.	Central nervous system; generalized.
44	Watts.	1932	F.	48	American.	Central nervous system; lungs.
45	Mook.	1932				Skin.
46	Johns and Attaway.	1933	M.	48	White.	Central nervous system; face.
47	Rogers and Jelsma.[47]	1933	M.	47	American.	Central nervous system.
48	Fitchett and Weidman.	1934	M.	18	Negro.	Central nervous system; generalized.
49	Cabot. Case 20241.	1934				Central nervoussystem.

patient recovered) and Jones's case[42] in the pharynx. In one case described by Sheppe[34] the lesion was apparently confined to the lung, and in Berghausen's case[43] the tongue was affected, with a possible unproved lesion in the lung. There have been only four or five cases of torulosis of the skin, and these have usually been associated with a generalized lesion. The cases of torulosis of the skin are those of Verse and Mook (described by Weidman[38]), Rappaport and Kaplan,[39] Urbach and Zach,[44] and possibly that of McGehee and Michelson,[41] since this lesion pointed on the skin surface.

Review of Australian Cases.

Four cases have been reported in the Australian literature. Although these were reported as cases of blastomycosis and the organism in two was named *Cryptococcus gilchristii*, in one *Saccharomyces tumefaciens* (Curtis), and was unnamed in the other, we feel that these cases were actually due to *Torula histolytica*.

The first of these was reported by Swift and Bull[48] in 1917 from Adelaide, South Australia.

The patient was a Chinese, aged fifty-three years, who was born in China, but who had lived thirty years in South Australia. He was admitted to the Adelaide Hospital on December 27, 1916, complaining of severe persistent headache, which had been present continuously for five weeks, and failing vision. There was no history of skin or lung trouble. The patient died suddenly on January 10, 1917.

Swift and Bull gave a detailed account of the cerebro-spinal fluid examinations and described a budding yeast-like organism which was present in the fluid. They classified this organism as belonging to the same group as *Cryptococcus gilchristii*. From the clinical history and their bacteriological findings it would seem that the organism was really *Torula histolytica*.

The second case was reported by Barlow[49] in 1922. At a meeting of The Medical Sciences Club of South Australia held in Adelaide on April 7, 1922, he described a yeast-like organism found in the cerebro-spinal fluid of a child aged five years. This child was born in Australia. It had been suffering from chronic meningitis for six or seven weeks. Barlow described the organism as being similar to that found by Swift and Bull, and classified it as

Cryptococcus gilchristii. Although full details of the case and the bacteriological investigations are not given, we assume that this organism was probably also *Torula histolytica*.

The third was reported by Williams[50] in 1922 from Melbourne.

The patient was a male, aged thirty-five years, who was admitted to the Melbourne Hospital on June 30, 1921, with the diagnosis of tuberculous meningitis. The patient showed signs of acute cerebral upset, namely, delirium, irritability, and lay curled up on one side. He was incapable of giving a history.

The fourth was reported by Badham[51] in 1922. He gives no account of the history or clinical findings of the case, but gives a preliminary report as to his investigations with the organism.

Although these are the only four cases published in the Australian literature, we have knowledge of others studied by Dr. Hayden, of Melbourne.

Present Case.[1]

Clinical History.

About five weeks before going to hospital, the patient, a woman aged twenty-six years, was seized with agonizing headache and vomiting. Examination showed that she had no fever and the pulse rate was about 80. She was ill for ten days—better one day and worse the next—when she suddenly developed right-sided facial paralysis and paralysis of the external rectus muscle of the right eye, together with diplopia and definite limitation of the field of vision. This condition gradually disappeared, vision becoming practically normal and vomiting and headache lessened. However, she then developed lethargy, from which it was possible to rouse her, but she immediately relapsed. A few days before admission to hospital she again manifested some squint and the right knee jerk was not elicited. She could stand up, but tended to fall over to the right side. She was now running a slight temperature, but did not complain of headache, and vomited perhaps only once daily. She was taking food fairly well.

On admission to hospital she was found to be semi-conscious at times, but at other times answered questions reasonably well. The pupils were somewhat dilated and reacted sluggishly to light. Definite papillitis was found in the left eye, and there was paresis of the right rectus muscle. She was quite unable to stand up. On the third day in hospital the patient died quite suddenly. No autopsy was obtained.

Laboratory Findings.

Blood examination revealed the following information:

Total red blood cells, per cubic millimetre	3,410,000
Hæmoglobin value	66%
Colour index	0·95

Differential count of white cells:

Band forms	14%	87%
Neutrophile cells	73%	
Lymphocytes		13%

In stained films the red cells showed no significant pathological changes, but some cells showed diffuse polychromasia. Blood platelets appeared to be increased.

A cerebro-spinal fluid examination was made. Lumbar puncture revealed that the cerebro-spinal fluid was under increased pressure. It contained blood and was quite transparent. Globulin was found to be slightly increased and the total protein was definitely increased. It contained 175 cells per cubic millimetre, the predominant cells being lymphocytes. There were a few polymorphonuclear cells and some large mononuclear cells containing a nucleus with a reticular structure. No tubercle bacilli were found and no organisms were noted in the films. The cerebro-spinal fluid failed to give the Wassermann reaction.

A further lumbar puncture on the following day gave a similar fluid, in which the cell count had increased to 235 cells per cubic millimetre and a differential count showed that there were 67% neutrophile cells and 33% lymphocytes.

Bacteriological examination: Cultures were made from the cerebro-spinal fluid on blood agar and gave a few greyish-white colonies, which grew to a considerable size. Microscopic examination of smears from these cultures revealed a yeast-like organism, which was at first suspected to be possibly a contamination. This was strongly contradicted by the finding of the organism in both samples of the cerebro-spinal fluid.

Just about this time the attention of Dr. A. H. Tebbutt had been drawn to a communication of Dr. Hayden, Saint Vincent's Hospital, Melbourne, on several cases of a torula infection of the central nervous system. Thinking that this organism might belong to that group, he handed over the cultures to us for further investigation.

Investigation of the Organism Isolated from the Present Case.

Morphological Appearances.

Gram's stain revealed a Gram-positive organism, which was ovoid or spherical in shape and which resembled the ordinary yeast in appearance. The organism showed true budding and often showed the presence of a small lateral stalk, to which was attached the bud. This appearance has been noted by previous workers and seems to be rather typical of the organism. Many of the organisms, however, showed the bud in close proximity to the parent body. No evidence of mycelium formation could be detected and there were no spores.

Cultural Findings.

Cultures were made on all the common laboratory media, and also on various forms of Sabouraud's medium. Profuse growth was easily obtained at 37° C. and also at room temperature. It was found that by placing the medium at 37° C. for twelve hours and then leaving it at room temperature the best and most rapid growth was obtained. The growth usually became profuse in forty-eight hours.

1. *Plain Agar.*—A profuse growth occurred at 37° C., the growth being well developed at forty-eight hours. The colonies were moist, thick and raised above the surface of the medium. They were discrete at first, but later became confluent. The colonies were white in colour.

2. *Blood Agar.*—On blood agar the growth was similar to that seen on agar except that it was more luxuriant.

3. *Broth.*—A moderate growth occurred at 37° C., the growth being well developed in forty-eight hours. The broth showed a heavy deposit at the bottom of the tube.

4. *Gelatine Slab.*—A marked growth occurred at room temperature in twenty-four hours. There was no evidence of liquefaction.

5. *Plain Sabouraud's Medium.*—A profuse growth was obtained in forty-eight hours either at 37° C. or at room temperature. The colonies were thick, raised and moist. They were white in colour, tending to become yellowish-white on older cultures. The

[1] We are indebted to Dr. C. B. Blackburn, Dr. R. E. Jeffries and Dr. A. H. Tebbutt for the clinical and pathological notes.

colonies were at first discrete, but later became confluent, forming a uniform growth on the surface of the medium.

6. *Sabouraud's Medium with Dextrose.*—The growth was similar to that obtained with plain Sabouraud's medium, but more luxuriant.

7. *Sabouraud's Medium with Maltose.*—A very luxuriant growth appeared more quickly than on any other medium. This medium appeared to be the best for growth.

Control cultures were made with a known *Torula histolytica* obtained from Dr. Hayden, of Melbourne, with an ordinary yeast, with *Monilia albicans* isolated from a case of thrush and with a monilia isolated from the sputum.

8. *Biochemical Reactions.*—Various carbohydrates were inoculated with the organism from the present case and with the control organisms already mentioned. No change was observed in any of the carbohydrates inoculated with *Torula histolytica* until after forty-eight hours. Later certain carbohydrates showed acid reaction and at the end of four weeks the reactions shown in Table III were observed. No gas was produced in any carbohydrates.

9. *Sporulation.*—The test for spores was carried out by the "plaster of Paris" method as described by Henrici.[52] Plaster blocks were prepared in large test tubes, inoculated and incubated at room temperature and at 30° C. No ascospores were seen.

10. *Mycelium Formation.*—Agar stab cultures were inoculated and were incubated at room temperature and at 30° C. for two months. At the end of this period there was no evidence of mycelium formation.

From the morphological and cultural characteristics of the organism and from the symptoms of the patient we concluded that we were dealing with an organism known as *Torula histolytica*, which had produced a leptomeningitis. To identify the organism further we carried out the following animal experiments. Inoculations were made with an emulsion of the organism from a forty-eight hour culture on Sabouraud's maltose agar in normal saline solution, the emulsion containing about $3{,}000 \times 10^6$ organisms per cubic centimetre. In some cases inoculations were also made using a similar suspension of a culture of monilia.

Experiments with Mice.

Mouse Number 1.

On October 11, 1933, a male adult white mouse (Mouse Number 1) was inoculated intraperitoneally with 0·5 cubic centimetre of a suspension of the organism in saline solution. The mouse showed no ill-effects until after five days. The animal then appeared to be ill and gradually became worse and died twelve days after inoculation on October 23, 1933.

At *post mortem* examination very little macroscopic change could be seen. There was some injection of the peritoneum with a slight intraperitoneal exudate. The organs showed nothing of note.

Direct smears and cultures on Sabouraud's maltose agar were made from the heart blood, the peritoneal cavity, the spleen, the liver and lungs. The direct smears, stained with Gram stain, all showed a Gram-positive yeast-like organism which showed true budding. The cultures all showed evidence of growth after forty-eight hours. The colonies were white, moist, thick and raised, and were similar to those seen on the original cultures of the organism from the cerebro-spinal fluid.

Microscopic sections were examined from the lungs, liver, spleen and heart.

Lungs.—Sections of the lungs were examined with the following stains.

(*a*) Hæmatoxylin and Eosin Stain.—With hæmatoxylin and eosin stain the lung showed considerable congestion and œdema. Throughout the lung there was lymphocytic infiltration. At the periphery, immediately beneath the pleura, the lymphocytes were scattered and spread out. Deeper in the lung substance, however, the lymphocytes were collected together in clusters, forming more or less concentrically arranged nodules. Within these nodules

Table III.

Organism.	Dextrose.	Lactose.	Mannite.	Saccharose.	Maltose.	Lævulose.	Sucrose.	Raffinose.	Dulcite.	Xylose.	Galactose.	Arabinose.	Adonite.	Dextrin.	Inosite.	Sorbite.	Amygdaline.	Mannose.	Inulin.
Known torula (Dr. Hayden)[1]	A.	—	—	—	—	A.	A.	Very Sl. A.	—	—	A.	A.	—	—	—	Sl. A.	—	A.	—
Organism from present case[2]	A.	—	—	—	—	A.	A.	—	—	—	A.	A.	—	—	—	Sl. A.	—	A.	—
Organism from present case[1]	A.	—	—	—	Sl. A.	A.	A.	—	—	—	A.	A.	—	—	—	Sl. A.	—	A.	—
Organism from present case (repeat)[1] ..	A.	—	—	—	Sl. A.	A.	A.	Sl. A.	—	—	A.	A.	—	—	—	Sl. A.	—	A.	—
Monilia albicans (from thrush)[1]	G.	—	—	—	G.	G.	—	—	—	—	G.	—	—	—	—	—	—	—	—
Monilia (from sputum)[1] ..	G.	—	—	—	G.	G.	—	—	—	—	—	—	—	—	—	—	—	—	—

[1] Read after four weeks at 30° C. [2] Read after three weeks at 30° C.
A.=acid; Sl. A.=slightly acid; G.=gas.

there was a certain amount of endothelial cell formation. No giant cells were seen. The blood vessels were engorged with blood and there was considerable perivascular lymphocytic infiltration. There was a wide collar of lymphocytes around the bronchi, which in some cases showed some catarrhal desquamation. Ovoid bodies resembling yeasts were seen throughout the lung tissue (see Figure II).

Figure II.

A section from the lung of a mouse showing the organism in the lung substance. Note the clear space surrounding the organism. Hæmatoxylin and eosin. ×600.

(*b*) Gram's Stain.—A section of lung tissue stained by Gram's method showed the presence of Gram-positive ovoid bodies within the lung substance. These organisms did not seem to bear any definite relationship to the lymphocytic collections. They occurred sometimes just beneath the pleura, sometimes in the interalveolar tissue, sometimes within the alveolar lumen and sometimes in the lymphocytic collections or in the perivascular collars. An occasional organism was seen within a blood vessel.

(*c*) Iron Hæmatoxylin.—With iron hæmatoxylin stain the organisms showed up very clearly as brownish refractile bodies. In nearly every case the organism was surrounded by a definite clear space.

Liver.—Sections of the liver were examined with the following stains.

(*a*) Hæmatoxylin and Eosin Stain.—With hæmatoxylin and eosin stain the liver showed considerable congestion and the blood vessels were engorged with blood. The liver cells were swollen and poorly staining, and there was a moderate degree of fatty degeneration. There was some lymphocytic infiltration in the portal canals. Scattered throughout the liver were tubercle-like nodules similar to the larger lymphocytic collections seen in the lung. These nodules showed a more definite concentric arrangement, however, and appeared to be limited by a capsule. The nodules were composed of lymphocytic cells and endotheloid cells. No giant cells were seen. Embedded in the nodules were ovoid bodies which showed eosinophilic staining and which resembled yeast-like organisms in appearance. These bodies were surrounded by a clear space. The nodules did not seem to bear any definite relationship to the portal canals, but occurred anywhere within the liver.

(*b*) Gram's Stain.—With Gram's stain the ovoid bodies within these nodules were seen to be Gram-positive. They were similar to the organisms seen in the lung. Similar organisms were also seen lying free in the liver substance and on the peritoneal surface of the liver. The organisms were always surrounded by a clear zone.

(*c*) Iron Hæmatoxylin Stain.—With iron hæmatoxylin stain the organisms could be seen as brownish refractile bodies. In this particular section organisms were seen within the lumen of some of the blood vessels.

Spleen.—Sections of the spleen were examined with the following stains.

(*a*) Hæmatoxylin and Eosin Stain.—With hæmatoxylin and eosin stain the spleen showed marked engorgement and endothelial proliferation in the lymph sinuses. No definite follicles could be made out.

(*b*) Gram's Stain.—Gram-positive ovoid bodies similar to those seen in the other organs were seen scattered throughout the splenic substance.

Heart.—Sections of the heart were examined with the following stains.

(*a*) Hæmatoxylin and Eosin Stain.—With hæmatoxylin and eosin stain the heart muscle showed marked toxic changes. There was engorgement of the blood vessels. No follicles were present.

(*b*) Gram's Stain.—One Gram-positive body was seen embedded in the heart muscle.

Mouse Number 2.

On October 11, 1933, a male adult white mouse (Mouse Number 2) was inoculated intraperitoneally with 0·5 cubic centimetre of a suspension from a forty-eight hour culture of *Monilia albicans* in normal saline solution. The mouse showed no ill-effects from the inoculation. It was kept for two months and was killed on December 20, 1933.

Cultures were made from the heart blood, liver, spleen and lungs on Sabouraud's maltose agar. These were sterile after fourteen days' incubation.

Macroscopic and microscopic examination of the organs revealed nothing of note.

Experiments with Rats.

On October 11, 1933, a male adult white rat (Rat Number 1) was inoculated intraperitoneally with 0·5 cubic centimetre of an emulsion of the organism in saline solution. The animal showed no ill-effects until seven days after the inoculation, when it became ill. The animal gradually became worse,

until at the end of fourteen days it appeared as though it would die. On the sixteenth day, however, it began to improve, and at the end of twenty-one days was apparently well again. It was kept for two months and killed on December 20, 1933. At *post mortem* examination no naked eye lesions could be detected.

Direct smears and cultures on Sabouraud's maltose agar were made from the heart blood, liver, spleen and lungs. The direct smears stained with Gram's stain all showed a Gram-positive yeast-like organism. The cultures after forty-eight hours' incubation showed a white, moist, raised growth resembling the growth of the original cultures from the cerebro-spinal fluid.

Microscopical sections of the organs were prepared and examined.

Lungs.—Sections of the lungs were examined with the following stains.

(*a*) Hæmatoxylin and Eosin Stain.—With hæmatoxylin and eosin stain the most noticeable change in the lungs was an intense peribronchial lymphocytic infiltration. This lymphocytic infiltration was seen chiefly around the larger bronchi, the bronchi being completely surrounded by a wide collar of cells. The majority of the cells were of lymphocytic type, but here and there were endotheloid cells. The bronchi around which this infiltration occurred showed some catarrhal desquamation. Throughout these masses of lymphocytic cells were seen yeast-like ovoid bodies surrounded by a clear space. Throughout the lung substance itself were numerous tubercle-like nodules. These nodules showed a concentric arrangement and were composed of lymphocytic and endotheloid cells. They varied considerably in size and appeared to be in various stages of formation. The larger nodules showed fairly large giant cells situated near the periphery of the nodule. The smaller nodules did not show giant cells, but showed a greater preponderance of lymphocytes. These nodules resembled very clearly the tubercles seen in a case of tuberculosis (see Figure III). Embedded in these nodules were yeast-like ovoid bodies which were surrounded by a clear space. Similar bodies were seen scattered diffusely throughout the lung, some being in the alveolar lumen and others in the interalveolar tissue. The blood vessels were engorged.

(*b*) Gram's Stain.—The ovoid bodies described above were seen to be Gram-positive.

(*c*) Iron Hæmatoxylin Stain.—With iron hæmatoxylin stain the ovoid bodies showed up clearly as brownish refractile bodies surrounded by a clear space.

Heart, Liver and Spleen.—The heart, liver and spleen were examined microscopically and showed engorgement and toxic change. No organisms were detected in these organs.

Experiments with Guinea-Pigs.

On October 11, 1933, an adult male guinea-pig (Guinea-Pig Number 1) was inoculated intraperitoneally with 0·5 cubic centimetre of an emulsion of the organism in saline solution. The animal showed no ill-effects following the inoculation. It was killed six weeks later, on November 25, 1933. At *post mortem* examination no macroscopic lesions were detected. Cultures on Sabouraud's maltose agar were made from the heart blood, liver, spleen and lungs. These were all sterile after fourteen days' incubation. Microscopic examination of the organs revealed nothing of note.

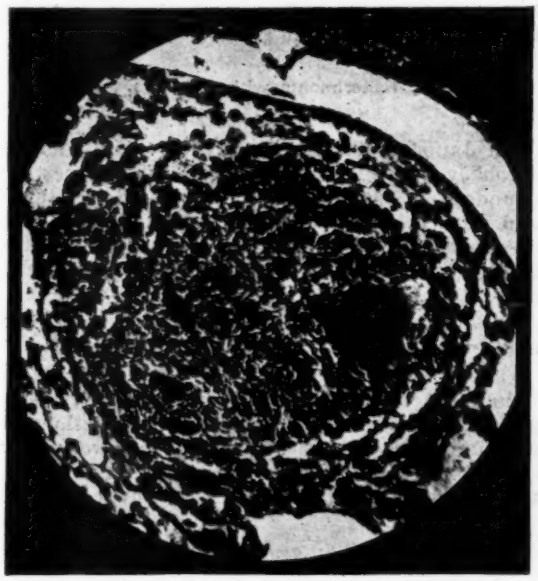

Figure III.

A section from the lung of a rat showing a typical "follicle" with giant cell formation. Hæmatoxylin and eosin. ×120.

On October 29, 1933, an adult male guinea-pig (Guinea-Pig Number 3) was inoculated subcutaneously with 0·5 cubic centimetre of an emulsion of the organism in saline solution. The animal showed no ill-effects and was killed on November 23, 1933. Cultures on Sabouraud's maltose agar were made from the site of inoculation and from the heart blood, the liver and the lungs. These were all sterile after fourteen days' incubation. Macroscopic and microscopic examination of the organs revealed nothing of note.

On October 11, 1933, a male adult guinea-pig (Guinea-Pig Number 2) was inoculated intraperitoneally with 0·5 cubic centimetre of a suspension of forty-eight hour culture of *Monilia albicans* in normal saline solution. The animal showed no ill-effects from the inoculation and was kept for two months. It was killed on December 20, 1933. Cultures on Sabouraud's maltose agar were made from the heart blood, lungs, liver and spleen. These were sterile after forty-four days' incubation. Macroscopic and microscopic examination of the organs revealed nothing of note.

Experiments with Rabbits.

On October 11, 1933, a male adult white rabbit (Rabbit No. 1) was inoculated intravenously with 0·5 cubic centimetre of an emulsion of the organism in saline solution. The animal showed no ill-effects from the injection and was killed six weeks later, on November 25, 1933.

Cultures on Sabouraud's maltose agar were made from the heart blood and from the lungs, liver and spleen. All cultures were sterile after fourteen days' incubation at 37° C. Macroscopic and microscopic examination of the organs revealed nothing of note.

Experiments with Monkeys.

Monkey Number 1.

On January 12, 1934, a *Macacus rhesus* monkey (Monkey Number 1), weighing 2,306 grammes (five pounds two ounces), was inoculated subdurally and intracerebrally with one cubic centimetre of a suspension of the organism in saline solution, strength about $3{,}000 \times 10^6$ organisms per cubic centimetre.

On January 12, 1934, five days later, the monkey appeared to be ill and would not take food.

On January 19, 1934, seven days after inoculation, the animal was *in extremis*. The pupils were equal and dilated, there was some head retraction, and the limbs were spastic. The monkey was chloroformed and killed. A *post mortem* examination was performed immediately and the following findings were recorded.

Macroscopic Findings.—The macroscopic findings were as follows.

Brain. The brain and *dura mater* were removed intact. The *dura mater* was tough and somewhat thickened. Around the site of inoculation, on the inner surface of the dura, there were numerous small, hard, yellowish, raised nodules resembling tubercles in appearance. These nodules were closely packed and extended away from the site of the needle puncture for about 5·0 centimetres (two inches). They were most numerous near the needle puncture and more scattered further away. The appearance of these tubercle-like nodules is shown in Figure IV. The brain itself showed some hæmorrhage around the site of the needle puncture. This was probably traumatic in origin. Adjacent to this the brain substance was very soft and presented a gelatinous appearance. There were small areas of hæmorrhage in this gelatinous part of the brain. No tubercle-like nodules were seen in the brain substance. The brain tissue remote from the site of inoculation showed no gross changes.

Spinal Cord. The spinal cord showed no macroscopic pathological changes. The *dura mater* was very carefully examined for nodules, but none were found.

Heart. The pericardial sac contained a considerable quantity of clear fluid. The heart muscle was slightly œdematous and showed some toxic change.

Lungs. There was no fluid in the pleural cavities. The lungs showed a patch of engorgement, but no other macroscopic change. There were no tubercle-like nodules in the lung.

Liver, Spleen and Kidneys. The liver, spleen and kidneys showed engorgement and toxic change, otherwise nothing of note.

Other organs were examined, but revealed no evidence of pathological change.

Bacteriological Findings. Cultures were made on Sabouraud's maltose agar from the heart blood, the brain and the spleen. A growth identical with that obtained from the original organism was isolated. Smears from these areas stained with Gram stain showed profuse Gram-positive ovoid bodies, similar to those found in the original cerebro-spinal fluid.

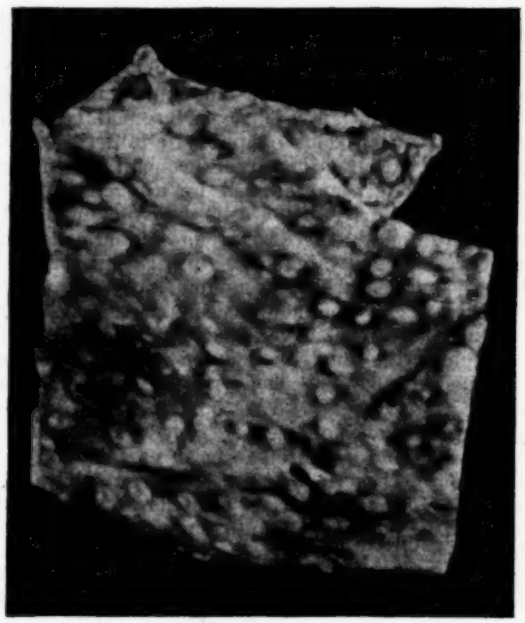

Figure IV.
Macroscopic appearance of the meninges of the brain of a monkey, showing the typical tubercle-like nodules scattered over the surface.

The organism recovered from the monkey was inoculated into the various carbohydrates and reactions identical with those given in Table III were obtained. The organism was also examined for mycelium and spore-production with negative results. It showed, however, true budding.

Microscopic Appearances.—The sections were stained with Mallory's stain. The findings were as follows.

Brain. Sections of the brain were taken through the gelatinous area noted macroscopically, adjacent to the site of inoculation. Along the needle track there was considerable hæmorrhage, which was probably traumatic in origin. Adjacent to this there was complete solution of the brain substance, with practically no inflammatory reaction. A few

scattered lymphocytes could be seen, but apart from this there was no evidence of inflammatory cells. In the gelatinous area the normal brain structure had completely disappeared. Large clear spaces were left, with here and there strands of structureless tissue running across them. In this area were numerous ovoid bodies resembling a yeast-like organism. These bodies were surrounded by a capsule, and some of them showed budding. The blood vessels of the brain were markedly engorged (see Figure V).

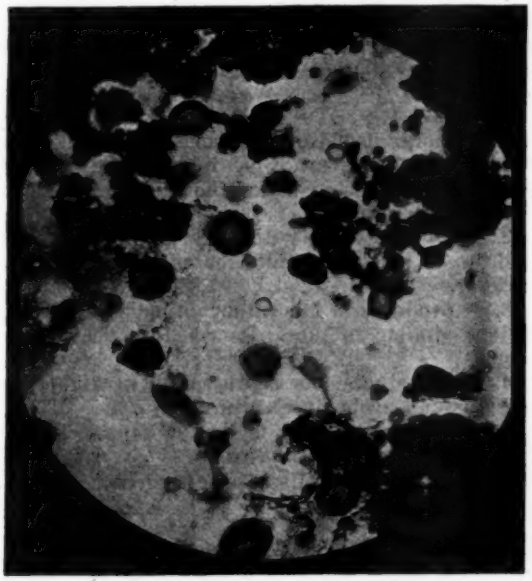

FIGURE V.

A section from the brain of a monkey showing the destruction of brain substance and the presence of numerous organisms. Mallory's stain. × 400 approximately.

Spinal Cord. Although macroscopically no evidence of infection was seen, there was definite microscopic evidence that infection had spread to the cord. The cord substance showed nothing of note beyond some engorgement of the blood vessels. The meninges, however, appeared thickened, and in the meninges were large collections of ovoid bodies similar to those seen in the brain. These bodies resembled yeasts and showed budding. Here again the absence of inflammatory reaction was a noteworthy feature (see Figure VI).

Lungs. There was intense engorgement of the lungs. There was some attempt at peribronchial lymphocytic infiltration, but this was not marked. Here and there in the lung substance were small collections of lymphocytic cells, but there was no definite nodule formation. Numerous ovoid bodies, lying in a clear space and showing budding, were seen throughout the lung, both in the alveolar tissue and in the alveoli themselves.

Spleen. The spleen showed intense engorgement. There was no definite nodule formation, but scattered throughout the splenic substance were ovoid bodies similar to those seen in the other organs. (The clear vacuolated space around these bodies, which is so typical, was well marked in the spleen.)

Monkey Number 2.

On December 5, 1934, a *Macacus rhesus* monkey (Monkey Number 2) was inoculated intravenously with one cubic centimetre of a torula suspension, containing 2,000 × 10^6 organisms per cubic centimetre. This monkey was chloroformed and killed on June 4, 1935, and its organs were examined for the presence of *Torula histolytica*. These organisms were not detected either by cultural tests or microscopic examination of sections of tissue.

Monkey Number 3.

A *Macacus rhesus* monkey (Monkey Number 3) was given 0·5 cubic centimetre of a torula suspension, containing 2,000 × 10^6 organisms per cubic centimetre, by hypodermic injection directly into the right lung on April 18, 1935. This monkey is still alive, and to date (July 19, 1935) has not shown any obvious signs of illness.

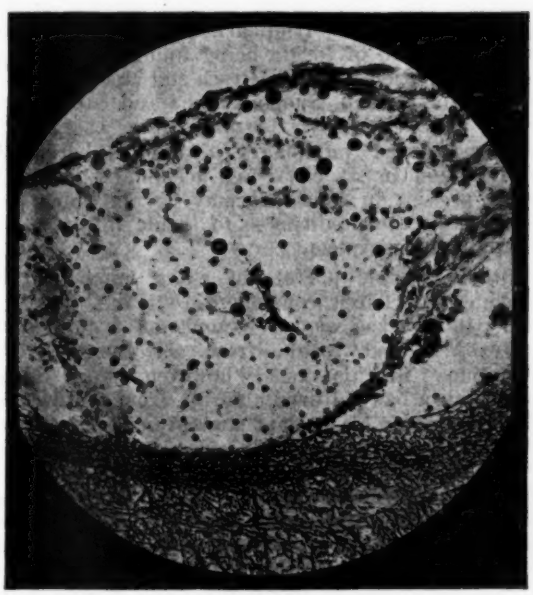

FIGURE VI.

A section from the spinal cord of a monkey showing the organisms present in the thickened meninges. Mallory's stain. Low power.

DISCUSSION.

The steadily increasing number of cases of this disease that have been reported indicates that infections by such organisms are either more frequent or that a correct diagnosis is made in a greater proportion of such cases. The latter appears to be the probable explanation.

The establishment of a final diagnosis is necessarily a laboratory function and, unless the possibility of such infection is borne in mind, the

microscopic appearances of preparations from the cerebro-spinal fluid may be overlooked and the presence of yeast-like colonies on cultivation may be attributed to contamination.

In the case recorded in this paper the clinical and laboratory findings have been in close accord with those described in the literature on this subject. The fermentation reactions reported by various observers have varied somewhat; but such results must be expected in this rather ill-defined group of organisms.

The attempts at monkey inoculation were successful only by rather gross intracerebral injection, but the pathological appearances obtained by this means corresponded very closely with the description of human lesions.

Clinically the recognized cases of torula infection usually involve the central nervous system, and the presence of meningitis most frequently calls attention to the condition.

With regard to the portal of entry, nothing has as yet been definitely proven. The general consensus of opinion is that infection occurs through the air passages and lungs, with further spread by the blood stream and the lymphatics. In support of this we have record of torula being found in the lungs of several patients (Hall, Hirsch and Mock, Pierson, Rusck and Farnell, Sheppe, Stone and Sturdivant, White and Williams). In many other cases there was associated pulmonary disease, tuberculosis being found in six cases and terminal pneumonia being present in four. It may be that such pulmonary disease predisposes to torula infection via the pulmonary system.

Other possible portals of entry must also be considered. Türck considered that the portal of entry in his case was the œsophagus. Alvarez[53] cultured a red torula from the tongue. The symptoms in his case, however, were local. Berghausen has cultured a torula from an ulcer on the tongue and his patient showed consolidation of the lungs and a palpable spleen. Jones found torula in scrapings from nodules in the pharynx. This patient showed systemic symptoms. Freeman and Weidman suggest the possibility of the tonsil as a portal of entry. Hranova[54] found a non-pathogenic torula in the tonsil of a young girl. Tonsillitis preceded the torulosis in three of the reported cases (Freeman and Weidman, Jones, Sheppe). The common finding of yeast-like organisms in the throat in normal individuals may prove to be of some significance.

In three cases the association of Hodgkin's disease with torulosis has been noted, and this has been fully discussed by Fitchett and Weidman. They demonstrated the presence of yeast organisms in enlarged lymph nodes from the head of the pancreas by animal inoculation. They bring forward the possibility of the intestine as a portal of entry. It is interesting to note that Tanaka[55] observed 10% saccharomyces in apparently normal mesenteric lymph nodes examined at necropsy.

Johns and Attaway[56] mention a case in which trauma over the scapula was followed by a deep mycotic infection which healed many months before the onset of the typical meningitis. In their own case the initial lesion proved to be a small superficial granuloma that developed from a razor cut.

Without careful examination the pathological appearances may easily be mistaken for tuberculosis, and it is more than possible that many cases have been diagnosed as such.

Our object in this paper has been to draw attention to the main facts of this interesting subject, the literature of which is not readily accessible, and it is expected that with careful examinations similar cases will be diagnosed.

Summary.

1. The organism isolated from the cerebro-spinal fluid of the patient was investigated bacteriologically and belongs to the group known as *Torula histolytica*.

2. The organism is pathogenic for mice and rats and, to a limited extent, for monkeys (*Macacus rhesus*).

3. It produces in laboratory animals pathological lesions similar to those described as occurring in man.

4. It produces a septicæmia in laboratory animals and probably also in man.

5. The case reported is one of lepto-meningitis caused by *Torula histolytica*.

6. A review of the literature is given.

Acknowledgements.

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To Dr. Hayden we are indebted for the culture of a similar case investigated in Melbourne.

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Reports of Cases.

AN UNUSUAL FRACTURE OF THE TALUS.

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THE following notes on a fracture of the talus, in which the mechanism of the fracture was of a very unusual nature, may prove of interest.

C.K., a carrier, was sitting on the ground with his feet resting on the ground, eating his lunch, after having placed a crate of fibro-cement on its edge against a wall. He was facing the crate when it tilted forwards, striking his right knee while the sole of his foot was resting on the ground. He was seen six days later with a greatly swollen right ankle, but with no damage to the skin. A radiographic examination disclosed no bony lesion of his patella, femur or tibia, but a comminuted fracture of the talus with three large fragments, namely, an anterior fragment which had been divided from the body through the neck, the fracture line running from above posteriorly and downwards towards the tip of the *os calcis*, and a medial and lateral fragment, of which the former was larger, and many smaller comminuted fragments in the intervening spaces. The medial fragment had been forced through the postero-medial portion of the capsule of the ankle joint and was pushing the plantar nerves and vessels towards the surface in a position posterior to the medial malleolus. The lateral fragment had been forced downwards and outwards into a position below and behind the lateral malleolus. Owing to the extensive dislocation and the wide separation of the fragments of the talus, they were removed and the foot was put up in plaster in a position of slight equinus and slight eversion.

The patient resumed his occupation, but about eighteen months later returned complaining of pain in the ankle, beneath the medial malleolus, which was worse on weight-bearing. A radiographic examination disclosed two small fragments of bone which were firmly embedded in fibrous tissue in the site originally occupied by the talus. These were removed and he has since resumed his work.

The chief point of interest is the extensive destruction of the talus by indirect violence through the femur, tibia and *os calcis* without any obvious injury to any of these bones.

Reviews.

DOMESTIC REMEDIES.

Gipsy Petulengro is the grandson of the old gipsy tinker immortalized in "Romany Rye" and "Lavengro". His mother was a Roumanian and from her he gained his knowledge of herbs and remedies which had been handed down by her ancestors for centuries. During the last thirty-five years Petulengro has lectured extensively on old Romany remedies and has published a booklet dealing with them.[1] The botanical kingdom is mostly concerned—roots, bark, leaves and flowers. The animal kingdom is not neglected, and we read that the fat of the hedgehog is good for deafness. Among inorganic salts we find bluestone and mercury oxide in their places. Petulengro repeats the old statement that most of the complaints occurring in certain countries can be cured by the herbs growing in the same places; and he makes the sweeping assertion that most of the medicines sold under proprietary names were originally given to the people in the form of Romany remedies. His one remedy for elevated blood pressure is the stinging nettle. With perfect justification he mentions that the vaunted remedy chlorophyll occurs naturally in plants which are habitually consumed. The remedies include many that are in common use, some even pharmacopœial. As examples we may quote couch grass (*Agropyrum*) for cystitis, periwinkle (*Vinca*) for diabetes, broom (*Scoparium*) for dropsy, rosemary as a hair stimulant, horehound for cough, *Geranium maculatum* (a tannin-containing plant) for infantile diarrhœa, and so on. Some are undoubtedly useful; but investigation has not confirmed the value of others. It is an interesting little book, but not of any special value.

COMPARATIVE ZOOLOGY.

There is no end to the production of text books, and as science advances this is not surprising. The new text book is often the only means by which the busy worker can keep up to date with new discoveries outside his own special and ever-narrowing field. "An Introduction to Comparative Zoology", by Whitfield and Wood, cannot, however, be said to help in this respect.[2] It is stated in the title to be a text book for medical and science students, and is obviously intended to be a first year text book only. The authors are entomologists and lecturers in zoology in the Soudan.

We are told that the book is an attempt to treat the subject of zoology from a comparative and evolutionary standpoint. We cannot agree that this has been achieved. This book is in no way different from a great many other first year text books of zoology which are not labelled "Comparative Zoology". Once again we have descriptions of the anatomy of familiar types—*Amœba*, *Entamœba*, *Euglena*, *Trypanosoma*, *Hydra*, and so on, the vertebrates being represented by the dogfish, frog and rabbit. There is one marked difference, however, in the number and selection of insect types. Three chapters are devoted to the Insecta, in one of which insects of medical and veterinary importance are discussed. This section is well done and excellently illustrated, and it is distinctly useful for medical students. But medical students will still have to await the ideal first year text book in zoology. In it there will be a more extended part devoted to genetics and the more fundamental aspects of zoology.

Where we find fault with this new addition to text book literature is in the number of inaccurate descriptions found in its pages. Some of these are due to loose statement, but others give quite incorrect data. Examples of the former are: "Fish arose first, gave rise to the frogs, which in turn gave rise to the rabbits." "Finding that the latter existence was more profitable, they [the frogs] evolved still further and became completely adapted to a terrestrial life, the final result being the rabbit."

Amongst the many errors are such statements as the following: (1) Speaking of excretion in the dogfish: "Urea in any quantity is toxic to the cells." (As it happens, over 2% urea is normally present in dogfish blood, and its presence there is absolutely essential!) (2) The posterior portion of the pituitary gland produces a hormone called "Pituitrin". (Pituitrin is an extract, not an individual hormone. Moreover, "Pituitrin" is actually a patented trade name for the original post-pituitary extract made by one particular commercial firm.) (3) "Brain cells were taken from a man in New York in 1912 and are still alive."

Such statements as these are not expected in a book of this kind and standing.

It remains to be said that the publishers have accomplished their part very well. The work is nicely got up, and the printing and general lay-out are excellent. There is a very complete index.

MATERNAL AND CHILD WELFARE.

"The Maternity and Child Welfare Movement"[1] is a more extensive contribution to the subject already dealt with by Dr. G. F. McCleary in his earlier work, "The Early History of the Infant Welfare Department". Maternal and child welfare are so closely interwoven that the method adopted by Dr. McCleary in this later work of dealing with the subject from both aspects provides a much more comprehensive view of the whole movement for the safeguarding of child life.

Much of the author's information was obtained through first-hand experience, and he has marshalled many facts which will doubtless be new even to the well-informed. Perhaps the most useful purpose served by the book (so far as the Australian reader is concerned at any rate) is the presentation of an unbiased and broad summary of the contributions made by the various pioneer workers in the movement. Some names popular with the general public are omitted altogether, while the labours of many others, previously unsung, are brought out into the light and their owners are given fair credit for their share in the building up of the present world-wide system of infant welfare.

One cannot help feeling that with such a wealth of material and a subject of such popular interest the matter could perhaps have been presented in a more interesting way. The book is, however, written in a simple style, which should make it a useful book of reference for the social worker as well as for the medical practitioner.

Notes on Books, Current Journals and New Appliances.

A LABORATORY MANUAL FOR PHOTOGRAPHY.

From Kodak (Australasia) Proprietary, Limited, comes a useful laboratory manual for photography. It should be of value to medical and dental radiologists, for it covers the whole range of their activities. The object of the brochure is to place before medical and dental practitioners authoritative information on the technique of handling X ray materials. The sections dealt with are as follow: "Intensifying Screens", "Handling of Films", "The Developing Room", "Developing Procedure", "Developing Dental X Ray Films", "Reduced Copies and Lantern Slides", "Clinical Photography", "Photography and Dermatology", "Infra-Red Photography", "Kodak Material and Accessories". The apparatus in each section is clearly described and the illustrations have been carefully chosen; there is no padding.

[1] "Romany Remedies and Recipes", by Gipsy Petulengro; 1935. London: Methuen and Company, Limited. Crown 8vo., pp. 58. Price: 2s. net.

[2] "An Introduction to Comparative Zoology: A Text-Book for Medical and Science Students", by F. G. S. Whitfield, F.R.E.S., F.R.M.S., and A. H. Wood, M.A., with foreword by R. Archibald, C.M.G., D.S.O., M.D.; 1935. London: J. and A. Churchill. Crown 4to., pp. 364, with illustrations. Price: 15s. net.

[1] "The Maternity and Child Welfare Movement", by G. F. McCleary, M.D., D.P.H.; 1935. London: P. S. King and Son, Limited. Crown 8vo., pp. 237. Price: 7s. 6d. net.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

SURGEONS AND SURGERY.

The George Adlington Syme Oration delivered by Professor F. Wood Jones before the Royal Australasian College of Surgeons at its annual meeting last March, and published in the July issue of *The Australian and New Zealand Journal of Surgery* under the title of "The Master Surgeon", was a contribution noteworthy from both the historical and literary points of view. But it was more than this—it was a call to surgeons to return to their high estate, to follow in mind and method, in outlook on life, the great masters who laid the foundations of the modern science and art of surgery. Surgery has become too easy. With aseptic methods and with anæsthesia ready at hand, anyone with a medical qualification may almost with impunity attempt to cure by mutilation. Non-medical people have not the fear of operation that was with just reason so common a century ago; and there are those who, bored by monotony or satiated by amusement, even go so far as to seek on the operating table a *divertissement*. In olden times there was little temptation to proceed to needless mutilation or to seek diversion at the hand of an operating surgeon. Surgery is used now much more than it ever has been, and every practitioner who undertakes surgical procedures would do well to heed the message written and implied in Professor Wood Jones's oration.

Professor Wood Jones described some of the attributes of a master surgeon. He dwelt upon the master surgeon's great humanity, "his pity and dislike for performing operations, that, however reluctantly, have been accepted as inevitable". He writes: "Paré, though in some respects a full-blooded old ruffian, startles us again and again with his intense pity for the wounded man, his dislike for inflicting pain, and his almost womanly solicitude for the stricken soldiers under his care." And so with other great masters of the craft. Present day operators might object that with anæsthesia and asepsis pain and risk of death are not so to be feared as they were, that intense pity need not therefore be shown, and that since cure by cutting is quick and perhaps simple, not to pursue it would be foolish. The surgeons of old time looked on the human body as something almost holy and not to be profaned; and those of today who are ripe in experience and mature in judgement know that every so-called cure wrought by taking away part of the body is a confession of failure. This is a truth that needs to be learned by every budding surgeon and, we have no doubt, by not a few of their older brethren. Another of the attributes of the master surgeon referred to by Professor Wood Jones (in view of his own calling he mentioned it first) was that all the great surgeons were great anatomists. They had need to be. There was no time for deliberate dissection, for the careful exposure and avoidance of vital structures; but they had to know exactly where structures lay, they had to be dextrous, quick and sure. The study of anatomy, however, was to them more than utilitarian—it was an attempt to slake their thirst for knowledge. What surgeon of the present time would get out of his bed at four o'clock in the morning to dissect beetles, as John Hunter did? Let us hear Professor Wood Jones:

> Nowadays we talk with pride of post-graduate studies. To these men pre-graduate and post-graduate days were all one. So long as they practised surgery they were practical students of anatomy. In their day this was an essential

condition of practice; and in truth anatomy may well be a life-long study for any man who would have the hardihood to tamper with the structure of the human body.

Today there lie before surgeons fields that were unknown to the old masters. The laboratory, with its microscope, its test tubes and its animal experiments, invites them to discover the cause of disease, to gauge its extent and to devise means for its cure! There is no doubt that, had the opportunity been given to him, the master surgeon of the past would have pursued laboratory investigations with the zest that he showed in every other direction. It is here that surgeons of today can, if they will, take up the torch where the master craftsmen laid it down. In many parts of the world this is being done, but not yet in Australia to any great extent. In any of the more important surgical journals that come from overseas will be found reports of experimental and laboratory investigations—perhaps one-half or more of the articles bear the title: "An Experimental Study." These investigations cover a very wide field; even biological chemistry and physics are included. It is absurd to pretend that a practising surgeon cannot find time for laboratory investigations. In the past surgeons have published in this journal the results of animal experiments and laboratory investigations undertaken in spite of busy hospital and private practices; but not very often. So long as surgeons in any country are content to put before the world writings that will "appeal to the average surgeon", and that may enhance their own reputations as manipulators and removalists, so long will the surgery of that country remain sterile. It is better to know why than to know how, to understand the cause of disease than to be able to cut it out with a knife.

Current Comment.

GIARDIA (LAMBLIA) INFESTATION.

Giardia intestinalis is a flagellated protozoon first described by Leeuwenhoek. Unfortunately it has received several synonyms which confuse the records of its investigation. These are *Lamblia duodenalis*, *Cercomonas intestinalis*, *Dimorphus muris* and *Megastoma enterioum*. Whether or not this protozoon is pathogenic to the human species is still a matter in dispute. Many are of the opinion that it is definitely pathogenic and produces clinical manifestations. In 1913 Melli-Leitao asserted that it caused diarrhœa and dysentery in children with mucus or even blood in the stools, but without griping or straining. During the Great War many hundreds of patients invalided to England from Gallipoli and the near East were infected with Giardia which was considered pathogenic. Porter, in South Africa, was strongly of that opinion, and she remarked on the difficulty of destroying the organism in the bowel. In the United States of America chronic intermittent diarrhœa, with abdominal pain and dyspepsia, has been attributed to Giardia and is stated to be very resistant to treatment. Labbé has described enteritis due to *Giardia intestinalis*. Musgrave reported from the Philippines that Giardia gave rise to intermittent diarrhœa, especially in children, and evoked a catarrhal condition of the intestinal mucosa. W. E. Boeck, however, is sceptical as to the pathogenicity of this organism and, although experimental observations suggest such pathogenicity, Boeck does not consider them conclusive. As fatalities are rare, not much help has come from autopsies.

R. M. Calder and R. H. Rigdon record a case of Giardia infestation associated with chronic inflammation of the gastro-intestinal tract[1]. A man, aged fifty-seven, a blacksmith and farmer, had suffered for sixteen years from intestinal discomfort with epigastric fullness and, later, intense burning feeling in the epigastrium. Occasionally nausea and vomiting followed up to thirty minutes after taking food. Alkalis at first relieved the symptoms, but later were ineffectual. During the two years before admission to hospital symptoms became very severe, with loss of weight, weakness and fainting attacks. For fourteen months prior to admission to hospital the patient had been able to retain only a very small amount of liquid food and eggs. Five days before admission profuse diarrhœa set in. On admission the teeth were seen to be affected with considerable caries and extensive pyorrhœa. The red blood cells numbered 4,250,000 and the white cells 19,550 per cubic millimetre. A blood Wassermann test gave no reaction and bacteriological examination of the blood revealed no organisms. X ray examination disclosed nothing abnormal in the stomach or duodenum, but there was definite spasm of the lower part of the descending and sigmoid colon. The stomach contents showed no free hydochloric acid even after histamine injection. The stools teemed with Giardiæ, at first motile, later only in encysted forms. Vomiting and diarrhœa became worse; food was refused and the patient died twelve days after admission. The temperature was at first normal, but later was elevated.

At autopsy the visceral and parietal pleura of both lungs were found to be bound together by old fibrous adhesions. Both lungs had areas of thickening of interstitial tissue. The gall-bladder

[1] *The American Journal of the Medical Sciences*, July, 1935.

contained brownish-black mucoid secretion teeming with Giardiæ, mostly in the vegetative stage. The lumen of the distal half of the transverse colon and of the descending and sigmoid colon was dilated. There was diffuse entero-colitis with atrophy and hyperæmia of the mucosa of the duodenum and large intestine. Part of the submucosa, especially in the colon, showed chronic inflammation. In some areas of the colon were superficial ulcers. The ulcer margins were ragged and hæmorrhagic and the bases slightly necrotic. The largest ulcer extended down to the muscularis. Some areas were covered by "diphtheritic" membrane. In the ulcerated areas no Giardiæ were found, but there were many Gram-positive and Gram-negative organisms, a Gram-positive diplococcus predominating.

C. Fairise and L. Jannin have reported an autopsy on a patient suffering from Giardia infestation and having chronic recurring diarrhœa. The necropsy showed fungating ulcers in the caecum and ascending colon. Calder and Rigdon suggest that, in their patient, possibly his grossly deficient diet may have been the primary cause of his symptoms and Giardiæ merely secondary invaders in an intestinal tract already injured as a result of chronic avitaminosis. The authors, however, make no reference to the possibility of tuberculosis, nor whether pyorrhœa might have had some ætiological connexion with the manifestations, nor what significance the hypochlorhydria may have had. The condition is certainly not proved to have been due to the Giardia infestation. Considering all the evidence, however, it would be wise to hold the Giardia as a pathogenic organism capable of inducing serious illness and even death. Whether Giardia may cause chronic cholecystitis is also a moot point. Boeck again is not satisfied with the evidence. The parasites have been found in the gall-bladder, but obviously such finding does not prove their pathogenicity, although such invasion might excite inflammation. In this connexion might be mentioned the fact that turkeys suffer from enterohepatitis called "blackhead". This disease is considered due to flagellate protozoon organisms. The only medicinal treatment received by Calder and Rigdon's patient was one injection of "Neoarsphenamine". Some investigators, however, are of the opinion that "Stovarsol" is efficacious in Giardia infestations.

THE BLOOD SUGAR.

When the estimation of the blood sugar content became a routine procedure in the diagnosis of *diabetes mellitus*, it was found that workers using Bang's method did not obtain the same results as those who used the method evolved by Lewis and Benedict. The significance of this was not recognized until it became known that the sugar content of arterial or capillary blood was not necessarily the same as that of venous blood. In Bang's method capillary blood is used, and in the method of Lewis and Benedict, venous blood. J. P. Bose has recently investigated the difference between the arterial and venous blood sugar content, and has made some interesting deductions.[1] Using the modified Folin-Wu method, he found that in healthy fasting persons the sugar content of arterial blood was slightly higher than or about the same as that of venous blood; in mild *diabetes mellitus* roughly the same relative values were found as in health; in "moderately severe" diabetes, either there was no difference or the value for venous blood slightly exceeded the value for arterial blood; in "severe" diabetes the value for venous blood was appreciably higher than the value for arterial blood. After the ingestion of fifty grammes of glucose by healthy persons the difference between the arterial and venous blood sugar content increased greatly. An average result was as follows: In the fasting state the arterial blood sugar content exceeded the venous by three milligrammes per 100 cubic centimetres; half an hour after the administration of 50 grammes of glucose the difference was 25 milligrammes; one hour after it was 35; one and a half hours after it was 14, and two and a half hours after, four. In mild diabetes the ingestion of glucose resulted in an appreciable increase in the arterial blood sugar content over the venous, but roughly only half that observed in health. In severe diabetes very little, if any, alteration in the relationship of arterial to venous blood sugar content occurred; generally, the greater the severity of the disease, the less the alteration. In some of the less severe cases the arterial blood sugar content, which was slightly lower than the venous in the fasting state, became slightly higher than the venous after the ingestion of glucose. In two remarkable cases the venous blood sugar content, which exceeded the arterial before the ingestion of glucose, exceeded it by still more afterwards. Bose concludes that "the magnitude of the difference between the sugar content of the arterial blood and the venous . . . after glucose ingestion is the best indication of the rate of glucose utilization in the tissues". In health the blood leaving the tissues contains less sugar than the blood flowing to them. In diabetes the power of the tissues to utilize sugar is diminished; therefore the venous blood becomes relatively richer in sugar. In some severe cases the venous blood sugar content is higher than the arterial, presumably because the tissues have lost even the power of retaining the sugar stored in them. Bose considers that an estimation of the difference between the arterial and venous blood sugar content gives far more information than the estimation of the capillary blood sugar content alone, or the glucose tolerance test as usually applied. Probably he is correct. His method may be of particular value in certain difficult or doubtful cases; but it will scarcely supplant current methods as a routine procedure.

[1] *The Indian Journal of Medical Research*, July, 1935.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Adrenalectomy in Rats.

Since active extracts of the adrenal cortex became available the mortality of young rats after adrenalectomy has acquired a new interest. Paul Schultz (*Journal of Physiology*, April, 1935) describes an improved operative technique by which, in a series of 77 animals, he has obtained a mortality of 100%. An inbred strain of white rats, obtained from the State Vitamin Laboratory, Copenhagen, was used in the experiments. Death occurred on an average after five to seven days. Autopsy was performed on all the animals. None of them showed any infection to be ascribed directly to the operation. There was almost complete absence of fat. No pneumonia, though frequently some congestion of the lungs, and no other gross changes in the organs which might be interpreted as a cause of death were found. Hæmorrhages in the gastric mucosa were almost a regular occurrence. Twenty-seven adrenalectomized rats were kept alive for twenty-one days after the operation by means of daily injection of cortical hormone. After discontinuance of the injections all the animals died, after an average of five to nine days. Some of them died even on the first or second day, and none lived longer than fourteen days after the last injection. The author points out that these results suggest the possibility of removing all cortical tissue by this improved technique, which is described and illustrated by a series of photographs. He admits that it is possible that he has been dealing with a strain of rats peculiar in having no accessory cortical tissue. But he considers it more likely that the tissue described by Gaunt and others as true cortical accessories may actually have been implanted fragments of cortical tissue detached from the adrenals at operation.

The Biological Significance of Copper and its Relation to Iron Metabolism.

C. A. Elvehjem (*Physiological Reviews*, July, 1935) reviews the present state of our knowledge of the occurrence of copper in plant and animal life, and of its biological significance. His conclusions are as follows. Copper is present in all living matter. The amount varies greatly in different organisms as well as in the different tissues of the same organisms. Two naturally occurring copper compounds are known: hæmocyanin, which is present in the blood of certain invertebrates, and turacin, which is found in the feathers of the South African bird, Turaco. Copper is of fundamental importance in the formation of hæmoglobin in all red-blooded animals. This does not mean that some of the other elements, such as manganese, zinc *et cetera*, are not essential to the living organism, but it does mean that they are not directly concerned in hæmoglobin formation. Milk is extremely low in copper as well as in iron, but a variety of natural foods will supply an adequate amount for normal nutrition. In the case of increased requirements, simple inorganic copper salts, together with iron salts, will allow adequate hæmoglobin formation. The difficulties encountered in obtaining clinical data as to the effect of copper in anæmia are, of course, very great. The most reasonable conclusion seems to be that available iron accompanied by small but standard amounts of copper should be used in all cases of anæmia showing reduced ability to form hæmoglobin. Copper is not concerned with the assimilation of iron, but with the transformation of the ingested iron into hæmoglobin. Recent studies concerning the mechanism of copper activity in the animal body deal mainly with the distribution of copper in different tissues, changes in the copper content of blood, the effect of copper on iron storage and utilization, and its relation to certain enzyme systems. It is well known that copper, like many other essential elements, is extremely toxic in large doses. However, the large number of laboratory animals which have been grown to maturity on milk mineralized with iron and copper demonstrates that copper in moderate doses produces no ill-effects. It is not now believed that hæmochromatosis is produced by chronic copper poisoning. However, it is well to keep in mind that, after any organism has received sufficient copper to meet the requirements for normal metabolism, additional amounts are of no value and may be of some harm. Copper is essential for plant growth, but its function aside from its association with chlorophyll formation is unknown. It also seems to be essential for yeast and other microorganisms.

The Effects of Hæmorrhage.

While the subject of blood volume after hæmorrhage has received sporadic attention since 1895, little or no work has been done upon the rapidity of the changes which take place. With the object of defining these changes more accurately than has hitherto been done, J. Douglas Robertson (*Journal of Physiology*, July 24, 1935) records observations on the blood and plasma volumes of cats after controlled hæmorrhage. The work afforded an opportunity for studying also the effects of hæmorrhage on blood sugar and on arterial blood pressure and for seeing whether correlation existed between the variations in blood pressure and those in blood volume. Cats were bled at an average rate of two cubic centimetres per minute per kilogram of body weight to varying degrees by the removal of 12%, 16·5%, 22%, 25%, 28%, 30%, 32%, and 37·5% of their original blood volume, and observations were made upon the consequent changes in the blood volume, the blood sugar, and the arterial blood pressure. The cats were anæsthetized by intraperitoneal injections of "Nembutal". A cannula in the carotid or femoral artery was connected with a manometer, while on the other side of the animal a cannula was placed in the carotid for the purpose of bleeding. A sample of blood was taken by cardiac puncture two or three minutes before hæmorrhage was commenced. The animal was then allowed to bleed to the desired amount. Immediately hæmorrhage was finished another sample was taken from the heart, and this was repeated at intervals of five minutes for the first quarter of an hour. Samples were taken half an hour and one hour after the end of the hæmorrhage. The blood withdrawn in each case was 1·5 cubic centimetres. Direct cardiac puncture was employed to insure obtaining a true specimen of the circulating blood. In the calculations it was assumed that the volume of blood before the experiment was 70 cubic centimetres per kilogram of body weight. Changes in blood volume were determined by hæmoglobinometry. The colorimetric acid hæmatin method of Newcomer was adopted. The plasma volume was determined from hæmatocrit values. These experiments showed that during a hæmorrhage there is a transfer of fluid from the tissues to the vascular system to make good the deficit in blood volume. Immediately the hæmorrhage finished, equilibrium in blood volume had practically been established, except in the most severe hæmorrhages (32% and 37·5%), where dilution of the blood continued until the animal died in ten minutes. This author shows that the withdrawal of plasma by the hæmorrhage was at the rate of 2·2 cubic centimetres per minute per kilogram of body weight, while the entrance of fluid from the tissues and fluid reserves to the vascular system during the hæmorrhage was 2·1 cubic centimetres per minute per kilogram. It can thus be seen how rapidly and perfectly blood volume is adjusted, the entrance of fluid from tissues to the vascular system being almost parallel with the loss of fluid from the vascular system by hæmorrhage. The return of the blood or plasma volume to normal must depend upon adequate fluid reserves in the tissue spaces, which in turn will depend upon an adequate fluid intake. If these reserves are sufficient, it has been demonstrated by these experiments that after blood loss the return of the blood volume to normal is almost immediate. The author also shows that there is an increase of the sugar content of the blood after a hæmorrhage; and the greater the hæmorrhage, the more pronounced is the hyperglycæmia. The source of the sugar is the liver, as hyperglycæmia does not take place if the hepatic vessels are ligated. As to the

effect on blood pressure, it is shown that when the hæmorrhage exceeded 30% of the blood volume, the blood pressure gradually dropped to reach zero in ten to twelve minutes, when the animals died. When the hæmorrhage did not exceed 30% of the blood volume, the blood pressure was lowest immediately the bleeding ended, then gradually rose after the hæmorrhage and reached equilibrium at a lower level than the original within ten minutes of the termination of the bleeding. There is no correlation between blood pressure and blood volume in these experiments, where, after hæmorrhage, an immediate transfer of fluid from the tissues to the blood stream has taken place. After a hæmorrhage, the hæmoglobin value is a very good index as to whether dilution has taken place, with its consequent effect in maintaining the previous level of the blood volume. In the presence of this dilution the above experiments suggest that the blood pressure gives a good indication, not of the blood volume, but of the blood loss.

BIOLOGICAL CHEMISTRY.

The Formation of Carbohydrate from Fat.

C. L. Gemmill and E. G. Holmes (*Biochemical Journal*, February, 1935) have presented results of experiments designed to determine whether fat can be converted into carbohydrate. Rats were given a diet of butter, salts and water for two or three days. The livers of these and normally fed control rats were used in the experiments. It was found that the respiratory quotient of the liver slices of the butter-fed rats was well below 0·7, the average value being 0·58. For the liver slices from the normally fed rats the respiratory quotient averaged 0·79. The carbohydrate content of the liver slices from the butter-fed rats showed a definite increase after shaking for three hours in a bicarbonate-Ringer medium at 37° C. It is contended that the lowering of the respiratory quotient and the increase in carbohydrate indicate that conversion of fat into carbohydrate occurred in the livers of the butter-fed rats, and that the same conversion may possibly occur in the normal liver. It was found that after one day on the butter diet the glycogen content of the liver fell practically to zero, but subsequently rose to a level of almost 1% on the fourth and fifth days of butter feeding.

Blood Coagulation.

H. Eagle, in a series of four papers, has discussed the results of experiments on several phases of blood coagulation (*The Journal of General Physiology*, March and July, 1935). He has advanced evidence of a quantitative nature to support the theory that calcium, a platelet factor (cephalin ?) and a plasma factor (prothrombin) interact to form thrombin, which reacts with fibrinogen to form fibrin. The amount and rate of thrombin formation were independent of the presence or absence of fibrinogen. After a variable latent period, thrombin suddenly appeared in large quantities, coincident with or immediately preceding deposition of fibrin, if fibrinogen was present. The amount of thrombin formed in a mixture of prothrombin, platelets and calcium was independent of the platelet or calcium concentrations and depended upon the amount of prothrombin used. Platelets or cephalin enormously accelerated the transformation of prothrombin to thrombin, and this was considered to be the physiological rôle of the platelets. Platelets were not found to contain significant amounts of prothrombin. The activity of the platelets was not species-specific. Calcium was recovered quantitatively from horse thrombin without affecting its coagulating activity, thus indicating that if thrombin was a calcium compound the calcium must have been present in minute concentration. Analysis also indicated that fibrin could not be a calcium-protein compound unless a molecular weight greater than 1,000,000 was assumed for fibrinogen. The experimental data did not permit a definite decision as to whether thrombin was an enzyme or whether it combined stoicheiometrically with fibrinogen to form fibrin, but the weight of evidence favoured the enzyme theory. The coagulation of fibrinogen by thrombin was found to be unaccompanied by a change in the hydrogen ion concentration in the free fluid. In hæmophilic blood the platelets were found to function normally and the prothrombin content was normal. Addition to hæmophilic blood of excess of platelets derived from either hæmophilic, normal human or animal blood accelerated thrombin production and effected normal clotting. The delayed coagulation of hæmophilic blood was considered to be due to an unexplained, retarded conversion of prothrombin to thrombin.

Magnesium Deficiency and Dental Structure.

H. Klein, E. R. Orent and E. V. McCollum (*American Journal of Physiology*, June, 1935) have studied the effect of a magnesium-deficient diet on the development of the teeth and their supporting structures in the rat. After weaning, rats were placed on a diet containing only 1·8 parts of magnesium per million. Control animals receiving the same diet *plus* added magnesium exhibited normal dental structures. In the animals given the magnesium-deficient diet dental changes became marked after one month and extremely marked after three months. The gingival tissue became a bulbous mass of smooth, whitish-grey tissue, deeply embedded in which were the molar teeth. The histological appearance of the gingivæ was abnormal. The molars were loosely embedded and could be readily lifted out with forceps. The proximal ends of the incisor teeth were widely separated by the gingival mass. The molar dentine was dense but striated, suggesting intermittent disturbance in calcification. The observed paradontal abnormalities were considered to be specifically caused by deficiency of magnesium.

Termination of Pregnancy in the Rabbit.

C. W. Bellerby (*Journal of Experimental Biology*, July, 1935) has shown that in the rabbit pregnancy can readily be terminated by intravenous injection with single small doses of an extract prepared from the anterior lobe of the pituitary gland. The effect is associated with atrophy of the existing *corpora lutea* of pregnancy, ovulation and the formation of hæmorrhagic follicles. All control animals were injected with saline solution and allowed to proceed to full term. All produced litters. The laboratory animals received injections at various stages of pregnancy and were killed on the third day after injection. In all cases abortion or resorption of the fœtuses occurred. In some cases the uterine contents were completely expelled. In others only placentæ were found in the uterus, and in such cases it was not known whether abortion had occurred or whether death of the embryos had been followed by complete resorption. In the majority the uterus contained fœtuses and placentæ in an advanced stage of resorption. In all the animals ovulation was induced and, from the appearance of the young *corpora lutea*, this had occurred at an early stage after injection. The functional life of the *corpora lutea* of pregnancy had presumably been terminated, as all these bodies had become white and non-vascularized and appeared to be in an early stage of atrophy.

Estimation of Alcohol.

A simple micro-method for the estimation of alcohol (ethanol) in biological materials is described by R. N. Harger (*Journal of Laboratory and Clinical Medicine*, April, 1935). Tissues are finely comminuted, acidified with tartaric acid and steam distilled. To avoid foaming with whole blood, distillation is carried out on a Folin-Wu tungstic acid, protein-free filtrate. Urine is distilled after dilution with water. In each case, to a portion of the distillate a known volume of a standard dichromate solution is added. Concentrated sulphuric acid is then added, and after standing for ten minutes the mixture is cooled. The heat developed on addition of sulphuric acid is sufficient to enable oxidation of the alcohol to acetic acid. The reaction mixture is titrated with an acid solution of ferrous sulphate and methyl orange. Normal blood and tissues show a small reduction by this method. Under the same conditions methanol is oxidized to carbon dioxide and water.

British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on October 2, 1935, Dr. Walter Summons, Vice-President, in the chair.

Clinical Observations on Blood Pressure.

Dr. Robert Southby read a paper entitled: "Some Clinical Observations on Blood Pressure and their Practical Application, with Special Reference to Variation of Blood Pressure Readings in the Two Arms" (see page 569).

Dr. Konrad Hiller, in opening the discussion, congratulated Dr. Southby. The paper was very valuable and Dr. Southby had arrived at some very important conclusions. The most arresting thing was the enormous differences in the pressures in the two arms. Dr. Hiller was unable to support or to dispute this observation, but, if Dr. Southby was correct, his observation was very important for those in practice and to insurance companies. If blood pressure estimations were made on the left arm, the companies would be satisfied, whereas if the same subjects were examined on the right arm, their proposals were likely to be rejected. Recently in London the Triennial International Conference of Life Insurance Medicine had been held and a *précis* of the proceedings had been published in *The British Medical Journal*. In the discussion on hypertension and the prognosis no one had referred to any difference on the two sides. He wondered what impression Dr. Southby's paper would have made if it had been read there. Since he had had the privilege of a preview of Dr. Southby's paper, Dr. Hiller had been taking blood pressure readings on the two arms of some of his patients, and already he had found a difference of twenty millimetres in a female patient, the systolic pressure on the right arm being 130 and on the left arm only 110 millimetres.

It seemed desirable in life assurance work that there should be unanimity as to normality, but it appeared difficult to get it; all the methods and standards were so different. Dr. Hiller was surprised that their readings were so much lower than one would expect; for instance, 140 millimetres at the age of sixty years. He recalled one patient of his who had had a systolic pressure of 160 millimetres at the age of sixty years and for ten or fifteen years afterwards and had remained perfectly well. He was convinced that standard blood pressure readings were too low in life assurance work.

The question as to what constituted early arteriosclerosis often arose in life assurance work. The blood pressure reading often was sent up by excitement, and he had noted that it might go up even higher at a second examination, sometimes from the same cause. Getting a standard of blood pressure was very difficult. The diagnosis of arteriosclerosis depended on a comparison of a reading with the normal reading for the individual and on the other clinical findings. When in young subjects the systolic reading was high, the diastolic reading became very important. If it was about 80 or even 90 millimetres, the condition was not early arteriosclerosis. In forming a prognosis a family history of high blood pressure or of deaths referable to raised tension was of great importance. From the life assurance aspect he agreed that it was much better to err on the safe side and to reject such an individual rather than to accept his proposal with a loading. On the other hand, in private practice one could watch the patients over long periods, and the immediate prognosis was helped by things going wrong that showed clinical evidence other than blood pressure readings; for example, if a patient had a renal condition without albuminuria, and albumin appeared, the prognosis became immediately bad. Dr. Hiller used to think that examination of the ocular fundi gave an indication as to the state of the cerebral vessels, but he was now afraid that this evidence was unreliable. A patient of his whom he had seen frequently over a period of fifteen years had suddenly developed behaviour changes which indicated impending trouble in the cerebral vessels, and though the appearances of the fundi remained perfectly normal, the patient had a hemiplegia within twelve hours of the examination of his fundi and died within three months. He felt, therefore, that examination of the eyes might not provide evidence of any prognostic value.

Dr. Hiller was inclined to agree with Dr. Southby that diastolic pressure readings were of great value in prognosis. He thought that a diastolic pressure over 100 millimetres was always a sign of arterial thickening and that, when the systolic pressure was high, the smaller the pulse pressure the worse was the prognosis. He would like to see more uniformity in the technique of making the readings. He took the systolic pressure when the first phase after the silence was beginning, and the diastolic pressure was taken either at the beginning of the fourth phase or at the end of it, when silence was reached. This variation made a difference of only about five millimetres as a rule, but he was not always able to recognize the fourth phase.

Dr. J. F. Mackeddie said that he, like Dr. Hiller, felt quite incompetent to deal with the question of disproportion that Dr. Southby had demonstrated. He had a guilty feeling that such chances of clinical observation had been missed by other people. He could not help congratulating Dr. Southby also on his explanation of the disproportion on the two sides, but he was puzzled to see an explanation of the evil significance of an increasing disproportion. If there was an anatomical basis for the disproportion, why should it have such an evil effect? It was not realized how important a standard diastolic pressure was and what a different outlook was required, depending on whether the pressure was over or under 100 millimetres, and yet this seemed to have been drummed into medical practitioners ever since their student days.

If Dr. Mackeddie wished to find out what normal blood pressure readings were, he did not think that he would go to insurance companies for the data. The range of normality for systolic pressures was from 90 to 160 millimetres, but the critical point to decide in a given case was what the blood pressure was doing; one estimation was of no value whatever. Dr. Mackeddie considered that a man who, while still lying on the couch, asked what his pressure was today, belonged to one of two classes: either the pressure would be subnormal, around 100 millimetres, or so-called high, about 160. That state of affairs was to a large extent the fault of the medical profession, because such pressures were probably quite harmless. It was eighty years since Claude Bernard had demonstrated the vaso-constrictor idea, and clinicians had known about the constrictor hormone for some forty years. They could apply these ideas in treatment, but they had been left without any efficient dilator and they had not got any further forward since they had been able to measure the pressure. Lots had been done in some ways: they did not use skimmed milk any longer or deprive the patients of protein or bleed them. But what had they instead? Dr. Mackeddie did not agree with those who told a patient with recurring nose bleedings that he needed a safety valve in his nose. If that was Nature's safety valve, it seemed to him that at times Nature went away and forgot to stop the safety valve. He had never found any harm in sealing up the crack in the nostril with a cautery, and he had found that it was a comfort to a patient to know that he did not need such a safety valve.

It was confusing to be discussing three types of raised pressure together, but in a general way Dr. Mackeddie considered that depressors could not be used with safety because no means of diminishing the constrictor action of the sympathetics were available. They had to turn to surgery for help in this problem, and, starting with Jonnescu through Royle to Adson, surgeons had attacked both the sympathetics and the hormone-producers. Adson might be considered a disciple of Royle and, though Royle had not been entirely successful in this work, repercussions from his work had led to extensive progress. Adson had gone so far as to remove sympathetics right along the

lumbar spine with only moderate influence on blood pressure, but, on cutting the nerve roots, he had brought blood pressure right down successfully. Dr. Mackeddie, in summarizing his views on treatment, said that he used hot applications with calomel. He thought that nitrites and iodides did not help much. He had dropped a lot of fads and had come to rely greatly on experimental physiology, from which a great deal more was still to be expected. Dr. Southby's valuable bit of work suggested care and anticipation, but just how it was going to lead to better treatment Dr. Mackeddie did not think that even Dr. Southby was able to say at the present stage.

Dr. H. Hume Turnbull said that he had not anything like enough figures to be able to discuss the main point in Dr. Southby's very interesting paper, but, based on the figures from the insurance companies, it appeared that with all young people blood pressures taken on the right arm would put them out, and on the left arm would put them in. No finer differentiation than five to ten millimetres in diastolic or ten to twenty millimetres in systolic pressure was worth anything. It was absurd to fix such an odd figure, say, as seventy-nine as a diastolic normal. Dr. Turnbull felt that as standards of normality the life insurance figures must be wrong, and he was not prepared to accept the table of percentages of life and death because no evidence had been put forward as to the cause of death and other relevant facts. End points were difficult and varied very much. A variation of ten to forty millimetres might be found when an individual's pressure was taken a few times by the same observer in any one day. However, it must be admitted that there was no other source for obtaining a large series of readings.

Dr. Turnbull remarked that a diastolic pressure of 110 millimetres meant a bad outlook in any case, and diastolic pressures of 140 or 150 millimetres were frightening, whether in the right or the left arm. He asked Dr. Southby to comment on the type of patient whose systolic blood pressure at rest gradually rose, though that taken when he had been walking about remained at about the same level. He instanced the case of a patient with a bed systolic pressure of 120, whose pressure when walking about was 210 millimetres. Four years later, in the same patient, though the reading was still 210 when walking about, the bed systolic had risen to 180 millimetres.

In conclusion, Dr. Turnbull expressed the opinion that, if Dr. Southby's observations were confirmed, it would be necessary to take fresh stock of the position from several different aspects.

Dr. S. O. Cowen joined with the previous speakers in appreciating Dr. Southby's initiative and the value of his undertaking. It was a most important piece of work and, of course, it would require confirmation. Dr. Cowen was unable to supply any personal data bearing on the difference of blood pressure on the two sides, but he questioned the admissibility of insurance figures as standards of normality. He had contributed some thousand erratic figures himself to insurance companies, but he hoped that they had not been included. It was difficult to get readings in a regular way. The answer to the question as to what constituted a patient's real blood pressure presented difficulties. He remembered a bank clerk, aged twenty-two years, who, when first examined by him, appeared to have a systolic pressure of 210 and a diastolic pressure of 140; it was not until the fourth or fifth session that he obtained the readings: systolic 120, diastolic 80 millimetres. Such experiences confirmed his view that correct figures were not at all often stated. The point of disappearance of the sound as that of the diastolic pressure was laid down because it was easier to note than the end of the fourth phase, and so was more likely to lead to uniformity. Dr. Cowen thought it was desirable to take three readings, deflating the cuff each time, and had often found a variation of twenty millimetres in the systolic records, though the diastolic records were more uniform, but might vary. He would like to refer briefly to a practical point which might depend on the emotional factor. He had noticed that when the cuff was being inflated the sound often disappeared at a figure which was lower than the one at which it reappeared on deflation. Dr. Cowen adopted an attitude of pessimism concerning the prognostic interpretation of raised blood pressure. He found it difficult to assess what a raised blood pressure meant. He instanced the utter unreliability from the prognostic point of view of relatively high readings in women around the menopausal age, for within five years the pressure readings might become normal again.

In conclusion, Dr. Cowen stated that a series of observations other than blood pressure records was necessary to the formulation of a sound prognosis; in spite of the specious simulation of exactitude that such figures presented, they could not rely on anything but a careful clinical review of the patient's illness.

Dr. W. Ostermeyer said that he was very interested in Dr. Southby's work and the discussion which followed the paper, because he had been present when Professor Martin had read a paper on the determination of arterial blood pressure in clinical practice at the Intercolonial Medical Congress in 1903. Dr. Ostermeyer also referred to a book by Janeway, published in 1904, in which was attributed to von Basch a statement that the radial pulse was selected for the estimation of blood pressure merely for convenience. After hearing Dr. Southby's explanation of the difference of the readings on the two arms and the experiments in hydrodynamics on which it was based, Dr. Ostermeyer was prepared to predict uniformity in femoral blood pressure readings. He would like Dr. Southby to carry out some similar investigations on temporal and on femoral pressures, and he would like him to rechart the pulse pressure readings. He thought that it would be demonstrable that the differences between the systolic and diastolic pressures would be fairly uniform. He believed that sinistrality was another factor that might account for higher blood pressures on the left arm than on the right. Dr. Ostermeyer had heard of apoplexy resulting from an acute fall in blood pressure. Hypotension required investigation as well as hypertension. It had been noted as long ago as 1904 that œdema vitiated blood pressure readings. Dr. Southby had associated the high readings with the urinary findings. Dr. Ostermeyer conjectured that viscosity of the blood might alter under certain circumstances, associated with albuminuria, and that this altered viscosity might lead to a rise in blood pressure.

Dr. L. S. Latham asked Dr. Southby if he could indicate the order in which he had made his observations on the two sides. If he had been in the habit of taking the pressure on the right side first, for instance, it might be of importance to allow psychological discount.

Dr. G. A. Penington said that he had recently noticed that the pressure on the right arm was greater than on the left, but he found usually that at about the fourth examination there was a tendency for the disparity to disappear.

Dr. Clive Fitts said that he had observed the blood pressure on both sides on a number of occasions, and a few bore out Dr. Southby's point. He had also taken a few femoral pressures and noted that they were somewhat higher. He had taken it on both sides by accident at first. Most of the patients had been elderly people with hypertension who suffered from atheromatous changes in the aorta. He had wondered what the explanation was for the difference to become more pronounced as the blood pressure rose. It was a fact that the arch of the aorta unbent as the blood pressure rose. The vessel lost its elasticity and even became tortuous. The formation of an aneurysm depended to a large extent on the amount of constriction of the ostia of the vessels where they left the aorta.

Dr. Southby, in reply, thanked those who had contributed to the discussion for the way in which they had received his paper and for the points of interest that had been raised. He did not feel inclined to agree with Dr. Hiller that a systolic pressure of 160 millimetres was within normal limits; he would consider that there was something wrong with such a patient. He did agree, however, that a familial tendency was significant of susceptibility to death from vascular conditions.

In reply to Dr. Mackeddie, Dr. Southby said that he was unable to offer an explanation for the increasing disproportion as the systolic pressure rose. He agreed that in normal circumstances individual readings between 90 millimetres and 160 millimetres were not of much value without other clinical results. Dr. Mackeddie's claim that no material advance in treatment had occurred in eighty years prompted Dr. Southby to relate the investigation of the blood pressures of certain American prisoners and their warders. Irrespective of age, previous mode of life and previous illnesses, all the prisoners seemed to have low pressures. This had been ascribed to the regular life and freedom from worry and anxiety and responsibility. On the other hand, the warders, who had to look after them and who were liable to dismissal if anything went amiss, had much higher blood pressure readings.

In reply to Dr. Turnbull, Dr. Southby agreed that small differences of two or three millimetres should not be stressed. They were within the limits of the personal equation in making the readings. He did not regard the difference between 75 and 80 millimetres in the diastolic readings as significant. He offered Dr. Turnbull an explanation of the rising bed pressure over a period of time by suggesting that it was likely that the difference between the bed pressure and the higher blood pressure on exertion indicated that such a person had a poor reserve in the cardio-vascular system and that as time went on this reserve tended to disappear.

In reply to Dr. Cowen, Dr. Southby stated that Dr. Cowen had hinted that it might necessitate a reshuffling of ideas if Dr. Southby's findings were confirmed. While he felt that this was over-stating the position, Dr. Southby thought that the higher readings should be those taken as a guide in prognosis and that, if it became necessary to make the choice, readings should be taken on the right arm. Dr. Southby was impelled by Dr. Cowen's remarks about the variation on inflation and on deflation to refer to the "auscultatory gap" which sometimes led to false readings. The sound might disappear and reappear at a much higher pressure and one might miss quite a high pressure. He instanced a patient in whom the sound was audible from 260 to 210 millimetres and disappeared, but reappeared at 140. This patient had died of uræmia a few weeks later. In reply to Dr. Ostermeyer, Dr. Southby mentioned that he had taken a few femoral pressures which tended to confirm his prediction. He agreed that the pulse pressure was very important. He had not gone into the question of the influence of left-handedness, but certain authorities said that there was no association between blood pressure readings and left-handedness. Dr. Southby assured Dr. Ostermeyer that the presence or absence of œdema in the arms had not interfered with his findings.

In reply to Dr. Latham's question, Dr. Southby stated that in the great majority of his cases the reading on the right arm would have been taken first, but in at least one-fifth the left one had been first. Two observers making simultaneous readings had noted the variation, and it did not make much difference which arm was used first.

In reply to Dr. Penington, Dr. Southby agreed that the psychological effect was a big factor that disturbed the accuracy of blood pressure findings, but, when the pressures were charted over a number of years, as his had been, this factor was eliminated. The effect of effort and exertion on blood pressure was interesting. When the Olympic Games were held in Berlin a large number of blood pressure estimations were made. They showed that with most of the younger athletes, the sprinters, middle distance runners, swimmers and cyclists, in good training, there was little departure from normal standards; but in the older group, the specialists, such as marathon runners, long-distance walkers, weight-putters and javelin throwers, the blood pressure average was distinctly higher. Dr. Southby was very pleased to hear what Dr. Fitts had said about his cardiac cases, and was interested in the statement that the pressure in the femorals was a little higher than in the brachial arteries; this would be accounted for by the presence of the extra column of blood.

MEDICO-POLITICAL.

Annual Meeting of the Delegates of the Affiliated Local Associations of Members with the Council of the New South Wales Branch of the British Medical Association.

The annual meeting of the delegates of the Local Associations affiliated with the New South Wales Branch of the British Medical Association was held at the British Medical Association House, 135, Macquarie Street, Sydney, on October 4, 1935, Dr. A. M. Davidson, the President, in the chair.

The following delegates were present: Dr. Kevin Byrne (Canterbury-Bankstown Medical Association), Dr. A. T. Roberts (Central Northern Medical Association), Dr. G. A. Buchanan (Central Southern Medical Association), Dr. G. N. M. Aitkens (Central Western Medical Association), Dr. A. M. Gledden (City Medical Association), Dr. G. J. Duncan (Eastern Suburbs Medical Association), Dr. W. F. Simmons (Illawarra Suburbs Medical Association), Dr. B. G. Wade (Kuring-gai Medical Association), Dr. A. G. Brydon (Northern District Medical Association), Dr. R. V. Graham (North-Eastern Medical Association), Dr. H. H. Lee (South-Eastern Medical Association), Dr. P. J. Markell (South Sydney Medical Association), Dr. E. A. Tivey (Warringah District Medical Association), Dr. J. T. Paton (Western Medical Association), Dr. W. H. Donald (Western Suburbs Medical Association).

The following members of the Council were present: Dr. G. M. Barron, Dr. George Bell, Dr. K. S. M. Brown, Dr. A. J. Collins, Dr. L. A. Dey, Dr. J. A. Dick, Dr. Hugh Hunter, Dr. W. K. Inglis, Dr. E. H. M. Stephen, Dr. Wilfred Vickers, Dr. A. S. Walker, Dr. G. C. Willcocks.

Dr. J. G. Hunter, Medical Secretary of the New South Wales Branch, was present.

The Editor of The Medical Journal of Australia, who sent an apology for his absence, was represented by Dr. W. L. Calov.

Welcome of Delegates.

Dr. A. M. Davidson, as President of the New South Wales Branch of the British Medical Association, welcomed the delegates in a brief speech.

Friendly Society Lodge Practice.

Removal of Names from Medical Officers' Lists.

It was resolved, on the motion of Dr. W. F. Simmons, seconded by Dr. A. G. Brydon:

> That a full list of names be submitted to the medical officer by the lodge secretary each quarter, and that lodge secretaries be requested to state the reason for the removal of names from the lodge list, either "deceased", "transferred", "left district" or "unfinancial".

Dr. W. F. Simmons pointed out that in Clause 5 of the Common Form of Agreement between Medical Officer and Friendly Society Lodge, the following words appeared: "The aforesaid list of members to be attended by the Medical Officer shall be delivered to him quarterly, that is to say, on the first day of January, April, July and October in each year." In Dr. Simmons's district the lodge secretaries did not supply quarterly lists; sometimes the medical officer did not receive a list for as long as eighteen months. This was highly unsatisfactory. He thought that there would be less cause for complaint if lodge secretaries complied with the rule. It would be a more satisfactory arrangement also if, when names were removed from a list, lodge secretaries informed the medical officer of the reason for their removal.

Visits to Patients during the Medical Officer's Accepted Half-Holiday and Evening off Duty.

Dr. W. F. Simmons moved and Dr. L. A. Dey seconded *pro forma*:

> That medical officers of lodges be under no obligation to visit members on the medical officers' accepted half-holiday and evening off.

Dr. W. F. Simmons said that the Illawarra Suburbs Medical Association had already submitted this proposal to the Medical Politics Committee of the Council, and the Medical Politics Committee had given a decision against it. In the district where Dr. Simmons practised it was the custom to charge fees for visits made during the accepted half-holiday and evening off. In some places, where there were two or more medical practitioners in partnership, a partner would make any necessary visits during the medical officer's half-holiday or evening off and would make no charge for these services. In Dr. Simmons's district the services were charged for whether they were rendered by a partner or by a colleague acting as the medical officer's *locum tenens* for the half-day or evening, as the case might be. Dr. Simmons pointed out that the lodges' dispensaries were closed during Saturday afternoon and Sunday. The lodges granted freedom from duty to their dispensers during this period, and they might extend a similar privilege to their medical officers.

Dr. Kevin Byrne said that some years ago he had had occasion to discuss this matter with Dr. R. H. Todd. Dr. Todd had stated that if a medical officer was away on business when an urgent call came to his house, on his return it was his duty to visit the patient requiring attention without delay; but if the medical officer was away on holiday, the patient could call in another medical practitioner, who was entitled to present an account for his services to the lodge; the lodge could then claim a like amount from the medical officer. This meant not only that the medical officer was virtually on duty all the time, but also that he was responsible for any expenses incurred by the lodge through his absence at any time. Dr. Byrne pointed out that medical practitioners were the only men working on a contract basis who were required to be on duty at all times.

Dr. Hugh Hunter said that it would be possible to effect the reform proposed by Dr. Simmons only by alteration of the Common Form of Agreement. He thought that any such alteration was inadvisable at present.

Dr. J. G. Hunter said that it was quite correct that medical officers of lodges were always on duty. It was the same in England. The medical officer must provide somebody to take his place if he was away at any time.

The motion was not carried.

Proposed Amendment of Clause 10 of the Common Form of Agreement.

It was resolved, on the motion of Dr. H. H. Lee, seconded by Dr. A. G. Brydon:

> That the Council be requested to have the model agreement amended by adding the following at the end of Clause 10: "Failure on the part of the Lodge Secretary to furnish the Medical Officer with the usual quarterly list shall in no way invalidate the claim of the Medical Officer, and provided such list has not been supplied for the quarter in question he shall be entitled to charge in accordance with the last list provided."

In proposing the motion, Dr. H. H. Lee remarked, among other things, that medical officers should be protected against defaulting lodge secretaries. A lodge secretary in a certain town had defaulted, and, because the list for the current quarter had not been supplied, the medical officer of the lodge had had no claim on the sum of £100 due to him. If no new list was supplied on the due date, the medical officer should be able to regard the list previously supplied as the true one, and act accordingly. Medical officers should not be penalized because of the incompetence, carelessness or dishonesty of some lodge secretaries.

Dr. Kevin Byrne thought that this was a matter requiring a legal opinion. He suggested that the amendment might invalidate the contract. If medical officers admitted treating patients whose names were not on the list, they admitted a breach of the contract.

Dr. G. C. Willcocks, Dr. A. T. Roberts, Dr. Hugh Hunter, Dr. A. S. Walker and Dr. L. A. Dey took part in the discussion.

Hospitals.

The Right of Patients in Intermediate Beds to Have Free Choice of Medical Attendant.

It was resolved, on the motion of Dr. P. J. Markell, seconded by Dr. W. F. Simmons:

> That intermediate patients in intermediate beds in every public hospital should have free choice as regards their medical attendant.

Dr. P. J. Markell said that he understood that any intermediate patient at a hospital under the *Public Hospitals Act* had the right of free choice of his medical attendant; but some hospitals were not under the Act. If the patient did not have the right to choose his own medical attendant, an anomalous situation might arise in which an honorary medical officer attended another medical practitioner's patient in a public hospital and could not accept fees from the patient. If honorary medical officers only were allowed to treat patients in intermediate beds, it would be detrimental to the honorary system. There would be a tendency for medical practitioners to seek positions on the honorary staffs of public hospitals with the object of obtaining the privilege of attending to intermediate patients. He liked to believe that honorary medical officers were altruistic in their motives and carried out their duties at public hospitals without thought of monetary gain. Dr. Markell mentioned a case that had recently come to his notice. A patient had been informed by his medical attendant that he required an operation for appendicitis. Later, the patient had been seized suddenly with acute abdominal pain and had been admitted to a public hospital and classified as an intermediate patient. An honorary surgeon had been called in urgently to perform appendicectomy. The honorary surgeon, realizing that the patient was not his, had then spoken by telephone to the patient's medical attendant, asking him if he desired to remove the patient. As by this time the patient had been prepared for operation, his usual medical attendant decided that it would be better for him to remain at the public hospital. Dr. Markell remarked that such a situation should never arise.

Dr. W. Vickers pointed out that Dr. Markell's proposition was part of the British Medical Association's hospital policy. Intermediate and private beds at public hospitals must be open. At the present time at the Royal Prince Alfred Hospital and the Royal Alexandra Hospital for Children there were intermediate beds, but no intermediate block with a separate operating theatre. The operating theatres at the Royal Alexandra Hospital for Children were at present fully occupied with the work of honorary medical officers, and for this reason the board of the hospital would not sanction the throwing open of the thirteen intermediate beds for surgical cases. The policy of this hospital would be for the beds in the intermediate block, when it was built, to be open to all medical practitioners. At present a compromise only was possible.

Dr. A. M. Davidson mentioned a case in which a medical practitioner had been unable to obtain admission to a public hospital of a patient suffering from acute appendicitis. Eventually the medical practitioner had arranged for a surgeon to perform the operation privately without charging a fee. The patient had been able to afford the private hospital fees and should have been admitted to an intermediate bed. The medical practitioner's complaint was that intermediate beds were all occupied at certain public hospitals, where his patient should have been treated, and honorary medical officers at these institutions were receiving payment for attending to patients of the same type as the one he had had to treat for nothing.

Dr. G. Bell, Dr. J. G. Hunter and Dr. A. G. Brydon also took part in the discussion.

Out-Patient Departments.

It was resolved, on the motion of Dr. G. J. Duncan, seconded by Dr. A. G. Brydon:

> That the attention of the British Medical Association Council should be directed to the fact that the British Medical Association's hospital

policy is not being carried out at all public hospitals in accordance with Clauses 9, 10 and 11 under Section VII, dealing with the out-patient problem.

Clauses 9, 10 and 11 of Section VII of the memorandum dealing with hospital policy read as follows:

> 9. Only such treatment should be given at the department as cannot in the best interests of the patient be obtained elsewhere under the usual arrangements as between private practitioner and private patient, or under contract arrangements.
>
> 10. Cases admitted for treatment at out-patient departments should be reviewed at regular intervals by the medical staffs.
>
> 11. No person, except in cases of emergency, should be accepted for treatment as an out-patient at a public hospital unless he brings a recommendation from a medical practitioner, a public clinic, or a government medical officer and such recommendation must be accompanied by a statutory declaration that the patient is unable to pay for private medical attention.

Dr. G. J. Duncan said that the Eastern Suburbs Medical Association viewed with grave concern the failure on the part of some hospitals to observe these clauses. He said that many patients who could well afford private fees were treated at the Prince Henry Hospital as out-patients and sometimes as in-patients also. Apparently this hospital was not concerned about fees. The Eastern Suburbs Medical Association had approached the Medical Secretary, and he had made representations to the Minister for Health with a view to bringing the Prince Henry Hospital under the control of the Hospitals Commission. The Eastern Suburbs Medical Association thought it was anomalous that the Government, which had appointed the Hospitals Commission of New South Wales, should not put its own hospitals under the control of the commission. This matter was not only of importance to the members of the Eastern Suburbs Medical Association; there were other State hospitals—Lidcombe State Hospital and Liverpool State Hospital. Dr. Duncan said that on the other hand the relations of the Eastern Suburbs Medical Association with the Eastern Suburbs Hospital had so far been quite happy.

Another feature was that the admission to the Prince Henry Hospital of patients who could really afford to pay might be responsible for keeping indigent patients out of the hospital through shortage of beds.

Dr. W. F. Simmons pointed out that the Prince Henry Hospital was not the only hospital at fault.

Dr. B. G. Wade also took part in the discussion.

Forwarding of Information by Public Hospital to Medical Attendant.

It was resolved, on the motion of Dr. G. J. Duncan, seconded by Dr. Hugh Hunter:

> That a patient sent to a public hospital for admission as an in-patient should be referred back, after treatment, to his medical attendant with a letter containing adequate information as to diagnosis and treatment.

Dr. G. J. Duncan said that it was difficult for the patient's medical attendant to get in direct communication with medical superintendents and other hospital authorities in order to obtain information concerning diagnosis and treatment. As a consequence the medical attendant was often left in the dark. In Dr. Duncan's experience the Royal Hospital for Women was the only hospital in the metropolitan area that regularly sent a report concerning patients that had been referred by medical practitioners.

Dr. A. S. Walker pointed out that it was difficult for an honorary medical officer at a public hospital to see that a report was sent or to send a report himself to the patient's medical attendant. It was inadvisable to give the report to the patient.

Dr. Kevin Byrne said that in his experience honorary medical officers were very courteous to general practitioners. Dr. Byrne thought that general practitioners should realize this and appreciate it. He agreed that it was desirable for the hospital to send a report to the medical attendant; but he thought that the medical attendant should also send a report to the hospital, dealing with progress and end-results.

Dr. H. H. Lee mentioned a patient who had suffered from epithelioma of the arm. Dr. Lee had removed the tumour; but some time later he had suspected a recurrence. He had referred the patient to a public hospital in the city with the request that a report be sent to him. The patient had had to make two visits to the hospital, paying a fee of ten shillings on each occasion as well as her train fares, which she had been ill able to afford. Eventually Dr. Lee had found it necessary to write for a report, which he had obtained at a fee of two shillings and sixpence.

Dr. W. F. Simmons described the system employed at the hospital in the district where he practised. Here the medical practitioner was always notified by the hospital.

Dr. A. J. Collins said that all would agree that the medical attendant should receive a report from the hospital; but there were difficulties. The matter was one for organization. The notification should not be the duty of the honorary medical officer, but of a special staff. It would be necessary to approach the Hospitals Commission. Dr. Collins quoted figures to show what a big undertaking it would be at an institution the size of the Royal Prince Alfred Hospital to send reports on all patients to their medical attendants.

Dr. E. A. Tivey, Dr. J. T. Paton, Dr. L. A. Dey and Dr. G. M. Barron also took part in the discussion.

Base Hospitals.

It was resolved, on the motion of Dr. G. A. Buchanan, seconded by Dr. J. T. Paton:

> That a conference be held in Sydney, open to all members of Local Associations, between representatives of the honorary medical staffs of proposed and established *A* base hospitals and the Hospitals Commission.

Dr. G. A. Buchanan said that there seemed still to be much confusion and uncertainty concerning the establishment of *A* base hospitals. They were still waiting at Goulburn for the Hospitals Commission to visit Goulburn to make its investigation in accordance with its promise. The members of the Central Southern Medical Association thought that if the Hospitals Commission would not visit them, the best thing to do was to arrange a meeting with the Hospitals Commission, in order to clear up the confusion.

Dr. J. T. Paton said that in Orange they were in a similar position to the medical practitioners in Goulburn. He thought that a conference was urgently necessary. There were several important points requiring discussion; for example: in the sale of a practice, presumably the position of honorary surgeon or honorary physician or other honorary position would go with the practice. The making of a general practitioner into a specialist in hospital work while he was still a general practitioner in his private practice was an insoluble problem. Dr. Paton suggested that it might be a good idea to create a position of honorary general practitioner for junior men. Later, these men could be classified as physicians or surgeons as they developed.

Dr. A. G. Brydon also took part in the discussion.

Registration of Hospitals under the Public Hospitals Act.

It was resolved, on the motion of Dr. H. H. Lee, seconded by Dr. B. G. Wade:

> That the Council of the British Medical Association be requested to interview the Minister for Health, drawing his attention to the fact that some hospitals are not registered under Section 35 of the *Public Hospitals Act* and to request him to exert his influence in the direction of compelling all hospitals so to register.

Dr. H. H. Lee pointed out that at Wollongong Hospital many patients were treated who could afford to pay private fees, and the honorary medical officers were not able to charge them for their services. No intermediate patients were taken at Wollongong Hospital. Wollongong Hospital was actually competing with private hospitals in the town. The medical practitioners had done all they could to improve the situation; now they could do no more. The registration of the hospital under the *Public Hospitals Act* was necessary.

Dr. J. G. Hunter said that the public hospitals at Wollongong, Cessnock and Kurri Kurri were probably the only country hospitals not registered under Section 35 of the *Public Hospitals Act*. He had spoken to the Hospitals Commission concerning them.

Dr. Kevin Byrne and Dr. A. M. Davidson also took part in the discussion.

Miscellaneous Matters.

Immunization against Diphtheria.

It was resolved, on the motion of Dr. A. G. Brydon, seconded by Dr. A. T. Roberts:

> That immediate consideration be given to the question of mass immunization against diphtheria in relation to the private practitioner.

Dr. A. G. Brydon said that the motion arose from the experience of medical practitioners in towns on the border of Queensland and New South Wales. In Queensland immunization against diphtheria was compulsory, and was carried out by private medical practitioners; consequently medical practitioners in border towns were sometimes required by the Queensland Government to immunize children on the Queensland side. At the same time, on the New South Wales side the medical practitioner performed no immunization, and if immunization was desired the New South Wales Government would dispatch a medical officer of the Board of Health to do the work. There was an apparent anomaly in that one government allowed medical practitioners to perform immunization while the other seemed to disapprove. It should be possible to show the public that the New South Wales medical practitioners were capable.

In reply to a question from Dr. H. H. Lee, Dr. Brydon said that "immediate consideration" meant consideration by the Board of Health.

Dr. J. T. Paton asked whether mass immunization had been considered by the New South Wales Government. He mentioned that there had been a protracted epidemic at Orange, where some private immunization only had been done.

Dr. J. G. Hunter said that if the work was to be done with the cooperation of medical practitioners, the Director-General of Health would require to know what fees would be suitable.

Dr. A. G. Brydon said that the fee paid by the Queensland Government was two shillings and sixpence for each immunization.

Dr. B. G. Wade pointed out that the method of immunization approved by the New South Wales Government would have some influence on the fee.

Far West Children's Health Scheme.

Dr. A. G. Brydon proposed and Dr. W. F. Simmons seconded:

> That the administrators of the Far West Children's Health Scheme be invited to cooperate more closely with the British Medical Association with a view to curtailing the amount of work done for this scheme by the general practitioner in an honorary capacity.

Dr. A. G. Brydon said that he spoke on behalf of one or two medical practitioners in western towns, who thought that some fee should be paid them for the work they performed on behalf of the Far West Children's Health Scheme.

Dr. G. M. Barron said that he was Chairman of the Far West Children's Health Scheme. Apparently there was some misunderstanding on the part of some medical practitioners in the far west. There had never been any atempt to foist honorary work on medical practitioners. No child was brought to Sydney for treatment unless a medical practitioner said he should be. The officers of the scheme were pleased to meet medical practitioners in every way.

Dr. Brydon, in reply, said that in no case had a medical practitioner begrudged the work he had done. Dr. Brydon mentioned that on occasions tonsillectomy and immunization against diphtheria had to be performed before children went to camp; it was for such work as this that the medical practitioners thought they might receive some payment. In view of Dr. Barron's explanation, and with the approval of his seconder, he withdrew the motion.

Fees for Attention to Police et cetera.

It was resolved, on the motion of Dr. J. T. Paton, seconded by Dr. A. G. Brydon:

> That the British Medical Association try to improve conditions of collecting fees due to doctors for services rendered to injured policemen, and for police cases.

Dr. J. T. Paton said that it seemed strange that police injured on duty did not come under the *Workers' Compensation Act* or some scheme for treatment. Police were instructed that, if injured on duty, they were to report to the nearest public hospital; that is, to obtain treatment from medical practitioners without payment. Also the police would not send for medical assistance for seriously ill persons or injured persons if they could avoid it. Unless the police themselves sent for medical assistance they were not responsible for medical fees.

Dr. K. S. M. Brown asked if there was any police regulation governing the police procedure in these cases.

Dr. J. G. Hunter said that not only a policeman but any public servant injured in the course of duty was entitled to free treatment at public hospitals.

Dr. E. A. Tivey and Dr. G. N. M. Aitkens also took part in the discussion.

National Insurance.

It was recolved, on the motion of Dr. Hugh Hunter, seconded by Dr. K. S. M. Brown:

> That it be a recommendation to the Council that the question of a national insurance scheme be considered.

Dr. Hugh Hunter said that medical practitioners must be prepared for national health insurance, and that it was advisable to have a proposed scheme ready. In nearly all countries, other than Australia and the United States of America, some scheme of national health insurance was in force. The recent remarks made by Dr. H. C. Dain had brought the matter to public notice, and something had to be done about it. Undoubtedly the general practitioner's conditions of practice had improved in England as the result of national health insurance; but would they be improved in Australia? There were many difficulties to be overcome before a satisfactory scheme could be instituted in Australia, and it was doubtful whether any scheme was required. Still, it seemed that the introduction of national health insurance was imminent, and therefore careful consideration by members of the medical profession was necessary. The Council desired to have the opinions of the Local Associations, and that was the reason for the motion.

Dr. K. S. M. Brown said that some scheme of national health insurance would shortly have to be considered. The Queensland Government was already introducing some scheme, with the exact details of which Dr. Brown was not familiar. The opinions of delegates of Local Associations were of importance, especially in relation to the possible effects of national health insurance on friendly society lodge practice.

Dr. J. G. Hunter asked the delegates to get the Local Associations to express their views on national health insurance to the Council.

Dr. A. M. Davidson said that no scheme would be proposed by the Council until it had been fully discussed by Local Associations and the views of Local Associations had been received.

Dr. A. M. Gledden said that while in England in 1934 he had found that the panel system was of great benefit to the medical profession; it increased the general practitioner's income and the sale value of his practice. Dr. Dain had failed to realize the big difference between the overhead expenses in England and Australia.

Dr. J. A. Dick, Dr. B. G. Wade and Dr. H. H. Lee also took part in the discussion.

Fees for Lectures to Nurses.

Dr. J. G. Hunter, Dr. A. G. Brydon, Dr. H. H. Lee, Dr. A. T. Roberts, Dr. J. T. Paton, Dr. G. A. Buchanan, Dr. B. G. Wade, Dr. A. M. Davidson and Dr. W. F. Simmons took part in a discussion on the payment of fees to honorary medical officers for lectures to nurses at public hospitals.

The "Handbook for Qualified Medical Practitioners".

Dr. K. S. M. Brown drew the attention of delegates to the wealth of information contained in the "Handbook for Qualified Medical Practitioners", issued during the year by the New South Wales Branch of the British Medical Association. He congratulated the Medical Secretary on his part in the compilation of the book.

Workers' Compensation.

The Treatment of Injured Workers by Salaried Medical Officers.

It was proposed by Dr. P. J. Markell and seconded by Dr. G. J. Duncan:

> That any member of the British Medical Association (New South Wales Branch) who is a salaried officer of a workers' compensation insurance company be debarred by the Branch from treating injured workers.

Dr. P. J. Markell pointed out that many employees were not aware that they had a free choice of medical attendant.

Dr. G. J. Duncan said that apparently insurance companies canvassed employers. He himself had had a notice from a company in which his employees were insured telling him to send any injured employees to the company's clinic and pointing out the various advantages of so doing.

Dr. A. M. Davidson expressed the belief that the way to obtain improvement was by propaganda among the workers themselves.

It was resolved, by way of amendment, proposed by Dr. G. C. Willcocks, seconded by Dr. H. H. Lee:

> That the Council of the New South Wales Branch of the British Medical Association approach the Government and ask it to endeavour to insure that the injured worker be advised of his right to select his own doctor.

Dr. J. T. Paton suggested that the various industrial unions might be circularized with a view to their informing their members of the workers' rights in regard to medical treatment.

Dr. G. J. Duncan suggested displaying notices in general practitioners' waiting rooms informing workers of their right of free choice of medical attendant.

Dr. J. G. Hunter, Dr. B. G. Wade, Dr. W. Vickers, Dr. E. A. Tivey, Dr. W. F. Simmons, Dr. Keith Inglis, Dr. G. M. Barron, Dr. K. S. M. Brown, Dr. Kevin Byrne and Dr. A. G. Brydon also took part in the discussion.

The Payment of Fees after the Death of an Injured Worker.

It was resolved, on the motion of Dr. G. A. Buchanan, second by Dr. Kevin Byrne:

> That the action of certain insurance companies in refusing payment in regard to insured workers who die soon after receiving injuries, be brought to the notice of the Council.

Dr. G. A. Buchanan said that there seemed to be some uncertainty about what the insurance companies were supposed to do in the event of the death of an injured worker. He wished to know what the medical practitioner could demand in such cases. Dr. Buchanan quoted a case in which a worker had died within a few days after the amputation of a limb. The insurance company had declined to pay the medical attendant because the patient had lived less than a specified number of days after the operation. It seemed to Dr. Buchanan that certain companies were interpreting the Act in their own way.

Dr. J. G. Hunter drew the attention of the meeting to the last paragraph on page 111 of the "Handbook for Qualified Medical Practitioners" issued by the New South Wales Branch, where it was stated that no provision was made in the *Workers' Compensation Act* for the payment of medical expenses in the event of the death of a worker as a result of injury arising out of and in the course of his employment. The medical attendant had to look for payment to the deceased worker's estate.

Dr. A. M. Davidson, Dr. K. S. M. Brown and Dr. J. T. Paton also took part in the discussion.

Fees for X Ray Examination.

It was resolved, on the motion of Dr. J. T. Paton, seconded by Dr. B. G. Wade:

> That the general practitioner making X ray examinations for patients insured under the *Workers' Compensation Act* be paid at the rate of 75% of full specialists' fees.

Dr. J. T. Paton pointed out that in some towns general practitioners doing X ray work got small fees only, as they were not recognized as specialists. Some insurance companies seemed to think they could pay these men whatever it suited them to pay.

Dr. J. G. Hunter said that this matter had been before the Council regularly. It was a difficult problem to solve. There were difficulties both when the general practitioner made an X ray examination and gave an opinion during the course of attention to an injured worker, and when he referred the injured worker to another general practitioner for X ray examination. The view of the Council was that, due regard being given to the overhead cost of an X ray plant, a fee of one and a half guineas was not unreasonable.

Dr. A. M. Davidson also took part in the discussion.

Luncheon.

The Council of the New South Wales Branch entertained the delegates at luncheon at the University Club.

NOMINATIONS AND ELECTIONS.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Claffy, Francis Patrick Christopher, M.B., B.S., 1933 (Univ. Sydney), 16, Boronia Street, Kensington.

Godfrey, Merle Franklin, L.R.C.P. and S., 1934 (Edinburgh), Sydney Sanitarium, Wahroonga.

Gunther, John Thomson, M.B., 1935 (Univ. Sydney), c.o. Lever's Pacific Plantations Proprietary, Limited, Gavutu, B.S.I.

Johnson, Alexander Skeffington, M.B., B.S., 1933 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Kingsley, John, M.B., 1933 (Univ. Sydney), District Hospital, Parramatta.

Yarad, Fred Callile, M.B., B.S., 1928 (Univ. Sydney), Balmain and District Hospital, Balmain.

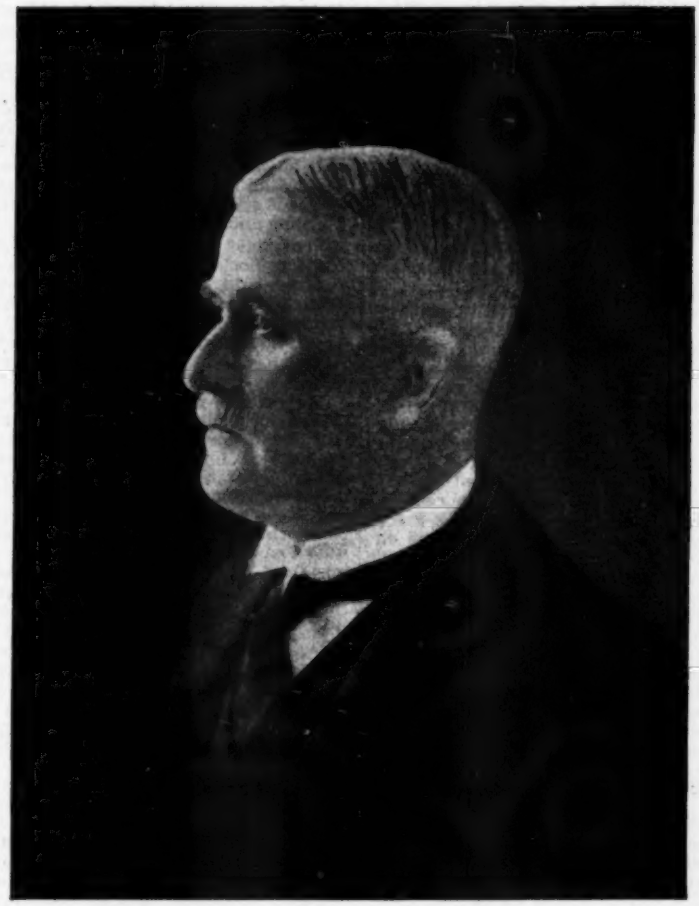
Obituary.

ALFRED AUSTIN LENDON.

We are indebted to Dr. W. A. Verco for the following account of the career of the late Dr. Alfred Austin Lendon.

Alfred Austin Lendon came to South Australia from London about fifty years ago. He assisted Dr. Davies Thomas for many years and ultimately took over Dr. Davies Thomas's practice. He gradually became a prominent medical man in the community and occupied quite a number of important and responsible positions.

At one time he was physician to the Adelaide Hospital. For many years he was one of the honorary officers at the Children's Hospital. Afterwards he was President of the Children's Hospital. After Dr. Way died he was appointed Lecturer in Obstetrics at the Adelaide University. He also filled the position of President of the South Australian Branch of the British Medical Association. He was President of the South Australian Branch of the Australasian Trained Nurses' Association. For many years he occupied the position of President of the District Trained Nursing Society. When he started with this society they were in debt; when he resigned his post, the society had a credit balance of several thousand pounds. This was due in great measure to Dr. Lendon's unflagging interest and industry in this very worthy cause. The society also spread its influence from the city and suburbs to the various country centres during his charge of their affairs. He was also President of the Numistic Society and took a great interest in all old and curious coins.



Several years ago he retired from practice and spent a great part of his time in delving into the early history of South Australia and Kangaroo Island, and read several papers on the subject. He also looked into the early history of the South Australian Branch of the British Medical Association, and wrote a history of the Medical School of South Australia, which is to have its jubilee this year.

He left a daughter, one son, Guy, prominent in medicine, and another, Alan, practising in surgery.

So passeth a very kindly and cultured gentleman, a man of much and varied knowledge, with an ever-alert mentality and of many good parts. Though gone from us, his memory and his works worthily remain.

Dr. F. S. Hone writes:

The death of Alfred Austin Lendon has removed one of the few surviving members of the group of medical men who fostered the life of the young medical school of Adelaide in the latter part of last century. He was one of the youngest of that band and in some respects the most picturesque. He came to Adelaide in 1883 with the degree of M.D., London. Youthful in years, he was still more youthful in appearance, and he retained the look of youth till late in life.

Almost immediately on his arrival he came into prominence through being dispatched to Bordertown in April, 1884, to investigate an outbreak of disease, which he proved to be one of smallpox. This was reported fully in the *Australasian Medical Gazette* at the end of that year, and his remarks on the confusion between chickenpox and so-called "native pox" (really *impetigo contagiosa*) are characteristic of the qualities of mind which he displayed throughout his later career.

His early partnership with the late Dr. Davies Thomas, from 1884 to 1887, gave him a fortunate introduction to medical practice in Adelaide, and the untimely illness of Dr. Thomas from 1890 until his death in 1893 left Dr. Lendon in possession of one of the largest and most fashionable medical practices in Adelaide. He was equally fortunate in attaining the rank of honorary physician at the Adelaide Hospital at an unusually early age. I have often thought how blessed we early students at the Adelaide Hospital were in having as our three clinical teachers in medicine men of such diverse ability and temperament as Dr. J. C. Verco, Dr. W. T. Hayward and Dr. A. A. Lendon. While Dr. Lendon did not claim the routine thoroughness in clinical investigation of Dr. Verco, or the extended clinical experience of Dr. Hayward, he had a more original mind, with a consequent stimulating effect on his students. In common with other students of that time, I owe much to his careful teaching and to his discussions in cases of doubtful diagnosis. His early association with Dr. Thomas had naturally interested him in the subject of hydatid disease, and he looked upon himself as the recipient of Elijah's mantle. This was shown by his collecting various papers by his late partner on the subject and publishing them in 1894 as the second volume of the treatise on hydatid disease, the first volume of which Thomas had published in 1884. To this volume he prefaced an appreciative memoir of Dr. Thomas. His interest in hydatid disease was invaluable to us as students, as he constantly kept it in front of us in his clinical teaching, and he followed this up by publishing in 1902 a volume

on "Hydatid Disease of the Lungs", in which he reproduced some clinical lectures he had delivered on the subject at the Adelaide Children's Hospital.

During those years he was also deeply interested in the subject of *erythema nodosum*, on which he held quite original views. His insistence, even in those days, on an infection as the basis of this condition was a great stimulus to thought, and made me follow with a deep personal interest later developments in the theory of its causation. His views were published in 1905 in a volume entitled "Nodal Fever", which makes interesting reading even now, with its record of his observations and actual experiments to try to prove the infectious nature of the disease. These two volumes demonstrate the originality of his mind and the clarity of his style, as well as his close observation of details, and his courage and pertinacity in endeavours to prove his case. The same qualities were shown in another field in later years in his devotion at the Adelaide Children's Hospital to the subject of "Ectopia Vesicæ". On this he wrote many short articles, and he was one of the earliest to transplant the ureters to open into the rectum as a preliminary to reparative surgery.

No one doubted his great ability in various directions nor his capacity for work; yet he never seemed to me to reach quite the eminence which his undoubted abilities deserved. One would say that he excited admiration for his ability and industry rather than that affectionate esteem in which some of his colleagues rejoiced. This must, I think, be attributed to certain foibles. He was widely read, both in the classics and modern languages, and took great pains in the preparation of his addresses; but he often concealed his earnestness over a cause he had at heart by a superficial cynicism; and he would sacrifice a serious address for a witty but flippant phrase or paragraph. As lecturer in forensic medicine in my student days, he invariably prefaced his course of lectures by a little discourse on the uselessness of lectures and a prohibition of any note-taking, and then would illumine a somewhat dry subject by his illustrations and his stimulating suggestions. In the wards he was fond of emphasizing the wealth of clinical material our small band of students enjoyed, and frequently informed us that he had never had the opportunity to examine a case of lobar pneumonia till he qualified. "Once I got within six students of examining one patient, but then she was so exhausted that the rest of us had to await another chance!" In later years, when he was lecturer in obstetrics, it was a legend that he started each year with the statement that the only qualification he had for the lectureship was that he had not attended a maternity case for ten years previously! Flippancies like these led to a suggestion of casualness or a lack of earnestness which was quite unwarranted. For again and again he proved himself capable of sacrifice for persons or causes that he held dear, as proved by his short editorial preface to Dr. Thomas's work which he edited, and by his well known refusal (in spite of great pressure at one time exerted in high quarters) to merge the South Australian District Trained Nursing Society (which was so close to his heart) into a larger Australian organization. I could imagine him writing a satire on "The Importance of being Flippant", or quoting with approval: "Better to reign in hell than serve in heaven." Certain it is that he loved to play the lone hand. How much this was all a pose no one can ever tell, but I am sure to the end of his days he delighted to be thought *l'enfant terrible*. During his last few years he turned his attention to the medical history of this State with great advantage to our medical literature. He showed the same painstaking industry as in his past clinical work in investigating historical records in the South Australian Archives Department, and even after his first attack of coronary thrombosis some three years before his death he filled us with admiration for his fortitude and industry in still pursuing these studies. Yet I understand his unexpurgated history has been deposited in the archives with instructions that it is not to be opened for fifty years, and even the expurgated edition prepared for the jubilee of the medical school has had to be considerably revised before publication.

None the less he had to his credit a great record of service, both private and public. In addition to the loyalty to his own family which characterized him throughout life, he adopted two babies—the Adelaide Children's Hospital and the South Australian District Trained Nursing Society, and the greater part of his professional life was devoted to the care and nurture of these babies until they reached adult life. Through one of those freakish elections peculiar to boards of management, he somehow, through no fault or shortcomings of his own, lost his reelection as honorary physician to the Adelaide Hospital in 1894. The "hospital row" was in full blast before the next triennial election was held, and after the Adelaide Hospital was reopened to medical students in 1900 he did not seek reelection, but confined his hospital activities to the Adelaide Children's Hospital. He had been an honorary medical officer at this institution since 1887, and when in later years the honorary staff was divided into physicians and surgeons he was appointed honorary surgeon. Against the wishes of all his friends who admired his work as a physician, he would persist in operative surgery, and still more in ambidextrous surgery!

In 1910 he resigned and was made honorary consulting surgeon and life governor of the hospital. He took great interest in the administration of the hospital, and while still an honorary medical officer was made vice-president. In 1902 he was appointed Deputy President of the Adelaide Children's Hospital Board, and until 1916 devoted much of his time and energy to building up its fortunes.

From 1898 to 1930, as President of the District Trained Nursing Society, he planned and worked with such energy that he built it from a small struggling body with nine branches to an organization with thirty branches and with nurses stationed as far apart as Mt. Gambier and Marree. In the meanwhile the working account had gone from £1,000 to £9,000, and he had built up an endowment fund of £20,500.

He was a member of the South Australian Medical Board for many years, and when he became President in 1917 was soon engaged in the passage of a revised *Medical Act*, which, for the first time in our State, introduced the principle of reciprocity in registration of medical practitioners from overseas, and only a few months before his death he was talking to me of improvements in registration which he thought were needed, even though he had given up office some years before. Nor must his services as chief medical officer to the Adelaide branch of the Australian Mutual Provident Society be forgotten. By his service in these various fields he showed how deep was his interest in all branches of his profession.

And so in 1928, when the Adelaide University Cancer Organization was in process of formation, he came to me and said how he would like to help. He was promptly made chairman of the organization and honorary director of the radio-therapy clinic, being appointed honorary consulting physician by the Adelaide Hospital Board in order to qualify him for that position. In that dual capacity he did much to put radio-therapy of cancer on a firm basis in Adelaide. He showed his characteristic enthusiasm over this new venture, arranging the details of the clinic's work, attending almost daily at the hospital, revitalizing the old cancer block, and creating an endowment fund. In 1931 he gave two university extension lectures on the subject of cancer, seizing the chance to expound to the public Sampson Handley's views on causation, to which he had become an enthusiastic convert. Shortly after this his first attack of coronary thrombosis occurred, in which his life was despaired of. With partial recovery he resumed his work on the medical history of the State, welcomed the chance of being the first President of the newly formed Historical Section of the local Branch of the British Medical Association, and, as he was housebound, asked for the first meeting to be held at his house and ably presided over it from his arm chair, chewing an occasional nitroglycerine tablet by the way! So, when eighteen months ago the jubilee celebrations of the Medical School were mooted, he promptly offered to write its history, and Sunday by Sunday consulted with the Vice-Chancellor over details—even to a fortnight before his

death. It is therefore fitting that his family have informed the Adelaide University Council of their decision to found a scholarship in his memory in the Medical School, to be awarded for medical research.

THOMAS HUGH BOYD.

We regret to announce the death of Dr. Thomas Hugh Boyd, which occurred on October 11, 1935, at Toorak, Victoria.

Correspondence.

PSORIASIS.

Sir: Your "Current Comment" of July 27, 1935. Schamberg, of Philadelphia, after much research suggested a positive protein metabolism in this disorder and obtained good results by limiting protein intake. He also forbade internal organs. German writers have suggested a relationship with diabetes, and have found that trouble and psoriasis combined and in families. In one family with whom I have been acquainted there are two cases of psoriasis in the same generation, there have been two cases of cholecystitis in different generations, one of whom had both conditions, and another in a later generation has diabetes. These writers advise a fat-poor diet. One of the psoriasis patients has entirely cleared the trouble away with diet, low fat, and the avoidance of internal organs, starchy foods and sugar. An acute attack of psoriasis can be brought on at will by the taking of these foods, the skin showing the lesions in two days or less. It appears to be elimination of a toxin by the skin. There are disturbances in the bowel and chest *et cetera* at the same time. In the issue of September 28 there is a note on vitamin *A* deficiency and minor degrees of vitamin deficiencies due to defective absorption from the alimentary tract, and it would appear that the cause of psoriasis will later be found to be due to metabolic causes rather than sepsis, although deficient functioning of organs like the appendix, and especially of the gall-bladder, with subsequent damage to the liver, cannot be excluded as prime causes. Other conditions, as boils, chilblains, asthma, paroxysmal rhinorrhœa, certain forms of rheumatism, scleroderma *et cetera*, appear to bear some relationship to the above. Further reports from Dr. McLaughlin will be looked for with interest.

Yours, etc.,

"Senex."

September 28, 1935.

Post Scriptum.—Blood sugar and blood cholesterin were not found in abnormal amount, and injections of liver extract intramuscularly were found experimentally to have no effect as compared with the crude product taken by mouth.

FIRE WALKING: A SIMPLE EXPLANATION.

Sir: The recent exhibition of fire walking by Kuda Bux, the Kashmiri magician, which took place in England has aroused a considerable amount of interest and a few observations on the point may lead one to a simple explanation of such a phenomenon.

Dr. Alexander Cannon, in his book, "Powers That Be", says in describing an exhibition by Dr. Tahra Bey, the Egyptian fakir:

> An operating table was brought on to the raised platform and Tahra Bey was laid thereon .... After a brief rest, his arms, face, neck and legs were cut and pierced with pins, knives and small bits of glass. A knife was plunged into his chest and withdrawn, whereafter he caused the blood to flow and to cease flowing at will. The wound quickly healed (within ten minutes to his own command) and his pulse was tested. It was shown that he had complete control of his pulse, for he could make it beat fast or slow at will ....
>
> One thing we noticed very definitely after the last demonstration had been given by Tahra Bey, and that was the complete absence of scars on the fakir's body, although he had been badly cut about and punctured with various weapons. The explanation given by him of this remarkable fact is interesting. Three things, he says, are necessary to the avoidance of scars. The first of these is a heightening of the pulse rate from the usual 60 to 70 beats a minute to about 120 to 135 a minute. The second is the temperature, and the third is the control of the micro-organisms which usually breed in a wound. No one of these conditions alone will have the desired effect, but the three things together will heal the wound in a remarkably short space of time.

These observations and phenomena thus prove that certain individuals can almost get complete voluntary control of the blood vascular system and there can be no doubt that they can send an increased amount of blood or a decreased amount of blood to any part of the skin.

There is a simple experiment which has a direct bearing on the subject in question, that anyone can try, namely, if an ordinary brown paper bag, from which the loose corner tags have been cut off, is filled with water, the water can be brought to the boil over an ordinary gas jet and the paper will not burn or scorch; this is due to the fact that the water inside the bag keeps the paper at approximately its own temperature, and as this temperature does not go above 212° F., no burning of the paper can take place.

Taking this experiment into consideration, one can see that if a sufficient stream of blood at the normal blood temperature is pumped through the skin of the sole of the foot, then the outer surface of the sole will remain at practically the same temperature as the blood immediately adjacent to it and under these conditions will not burn. We know that these fakirs have the power of increasing the blood supply to any part of the skin and, in this instance, to the soles of the feet. I think this is a simple explanation as to why the skin, and even a piece of sticking plaster attached thereon, did not burn when coming into contact with the red hot embers.

What should give us cause for wonder is not the fact that the skin did not burn, but the fact that a human being should be capable of such mental development as to be able completely to control his sympathetic nervous system. Might not there be in the principles underlying the development of such tremendous nervous control some hope for the alleviation of our fear-ridden, nervous, materialistic civilization?

Yours etc.,

D. W. H. Arnott.

143, Macquarie Street,
Sydney,
October 9, 1935.

Books Received.

PHYSIOLOGY IN MODERN MEDICINE, by J. J. R. Macleod, M.B., LL.D., D.Sc., F.R.C.P., F.R.S., assisted by P. Bard *et alii*; Seventh Edition; 1935. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay. Royal 8vo., pp. 1186, with 297 illustrations, including seven plates in colour. Price: 52s. net.

DISEASES OF THE THYROID GLAND, by A. E. Hertzler, M.D., with a Chapter on Hospital Management of Goiter Patients, by V. E. Chesky, M.D.; Third Edition, entirely rewritten; 1935. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay. Royal 8vo., pp. 348, with illustrations. Price: 45s. net.

Diary for the Month.

OCT. 31.—South Australian Branch, B.M.A.: Branch.
OCT. 31.—New South Wales Branch, B.M.A.: Branch.
Nov. 1.—Queensland Branch, B.M.A.: Branch.
Nov. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 5.—Tasmanian Branch, B.M.A.: Council.
Nov. 6.—Western Australian Branch, B.M.A.: Council.
Nov. 7.—South Australian Branch, B.M.A.: Council.
Nov. 8.—Queensland Branch, B.M.A.: Council.
Nov. 12.—Tasmanian Branch, B.M.A.: Branch.
Nov. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 13.—Victorian Branch, B.M.A.: Branch.
Nov. 19.—Tasmanian Branch, B.M.A.: Council.
Nov. 19.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 20.—Western Australian Branch, B.M.A.: Branch.
Nov. 20.—Victorian Branch, B.M.A.: Clinical Meeting.
Nov. 21.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 22.—Queensland Branch, B.M.A.: Council.
Nov. 23.—Victorian Branch, B.M.A.: Country Branch Meeting (Horsham).

Medical Appointments.

Dr. E. J. Ryan has been appointed a Member of the Medical Board constituted in pursuance of the provisions of *The Workers' Compensation (Lead Poisoning, Mount Isa) Act of* 1933, Queensland.

* * *

Dr. R. St. C. Steuart has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, to be Certifying Medical Practitioner and also Medical Referee at Melbourne, Victoria.

* * *

Dr. A. M. Myers (B.M.A.) has been appointed Resident Casualty Medical Officer, Adelaide Hospital, South Australia.

* * *

Dr. M. P. Susman (B.M.A.) has been appointed Honorary Consulting Thoracic Surgeon, Prince Henry Hospital Auxiliary, New South Wales.

* * *

Dr. W. M. Ada (B.M.A.) has been appointed Government Medical Officer at Delegate, New South Wales.

* * *

Dr. G. Trahair (B.M.A.) has been appointed Medical Officer, Department of Mental Hospitals, New South Wales.

* * *

The undermentioned have been appointed Resident Medical Officers at the Adelaide Hospital, South Australia: Dr. J. M. Pedler, Dr. F. F. Heddle, Dr. F. Flaherty.

* * *

Dr. I. W. McNaught (B.M.A.) has been appointed Government Medical Officer at Werris Creek, New South Wales.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii, xix. and xx.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Pathologist and Bio-Chemist.

PUBLIC SERVICE BOARD, ADELAIDE, SOUTH AUSTRALIA: Director of the Government Laboratory of Bacteriology and Pathology.

QUEEN VICTORIA MEMORIAL HOSPITAL, MELBOURNE, VICTORIA: Medical Superintendent (female).

RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Honorary Relieving Physicians.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, BRISBANE, QUEENSLAND: Honorary Officers.

THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Physician, Part-Time Pathologist.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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